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Virginia MEDICAL MONTHLY

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CONTENTS

Guest Editorial. William R. Jordan, M.D., Richmond, Va.---- 557

The Future of Medicine in Virginia. M. Pierce Rucker, M.D., Richmond, Va. ---------- 559

The Advantages and Limitations of Hormone Therapy in Benign and Malignant Prostatism. Linwood D. Keyser, M.D., Roanoke, Va. ---------- 564

Meningitis in Infancy. Wm. B. McIlwaine III, M.D., and Herman Farber, M.D., Petersburg, Va. ---------- 574

Pre-Operative Study of the Malformed Nose. Wm. Lawrence Gatewood, M.D., Richmond, Va. ---------- 579

Etiological Factors in Pancreatitis. John B. Floyd, Jr., M.D., Lexington, Ky. ---------- 583

Optochiasmatic Arachnoiditis Report of a Case. Morgan B. Raiford, M.D., M.Sc., (Med), Atlanta, Ga. ---------- 586

The Treatment of Bacterial Endocarditis: Review of Twelve Cases. Robert K. Williams, M.D., Baltimore, Md., and Kinloch Nelson, M.D., Richmond, Va. ---------- 589

The Treatment of Post Thrombophlebitic Sequelae in the Lower Extremities. Eugene L. Lowenberg, M.D., F.A.C.S., Norfolk, Va. ---------- 594

Continued on page 6.

November, 1949

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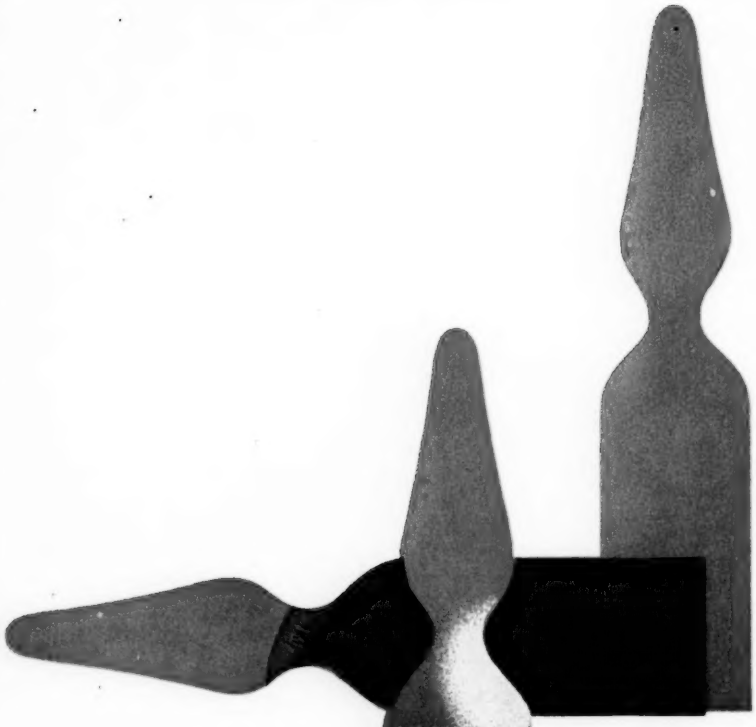

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GUEST EDITORIAL

The Problem of Diabetes

"OF all the common diseases diabetes goes undiagnosed most often and this in spite of the fact that no elaborate laboratory procedure, no extensive study and no expensive equipment are needed to make this diagnosis." A professor of medicine made this remark to me just recently.

It is amazing how often and how long a patient can suffer with diabetes before the diagnosis is made. It is not uncommon to see the disease progress to its final stage of coma without being diagnosed. Even more common is the patient, who has been treated for diabetes and who has remained in good health, only to fall sick two or three years later without diabetes being suspected as the cause of his new illness. At times the correct diagnosis is made only because the patient is admitted to a hospital where in the course of time the routine laboratory work discloses the diagnosis which until then has not been considered. Even in the modern hospital an unconscious patient may stay there for hours in diabetic coma without treatment until the laboratory reports the routine urinalysis. This is especially astonishing in view of the fact that a simple urine test costing less than five cents and requiring less than five minutes will yield the diagnosis in the majority of diabetics. Of course, such an omission in diagnosis of the really sick person is rare compared to the less acutely ill person. In the latter, the routine urinalysis may be taken long after the patient has taken food and shows no sugar. Remembering this, we should make an effort to examine the postprandial urine and take the blood sugar an hour after a real meal rather than after the patient has fasted.

In Dr. Joslin's home town of Oxford, Massachusetts, a survey was made of about 75% of the inhabitants. Approximately as many unknown diabetics were detected as those in whom the diagnosis had already been made. The conclusion one may draw is that, for every diabetic we treat, there is another who has not been discovered and who can obtain no such benefit. This diagnostic omission has probably been a factor in the Federal government's desire to carry on such surveys. One such survey is now being planned for Richmond. If the doctors of the State will solve this problem themselves, there probably will be less governmental activity of this sort. The American Diabetes Association has instituted a case finding program which has brought to light many an unknown diabetic, and similar private effort should be encouraged.

Other than the above, the most neglected phase of the handling of this disease in Virginia seems to be inadequate training of these patients. The son of a former patient of mine stated the other day that the last twelve years of his father's life could have been quite different and less burdensome if he had known at the onset of his diabetes what he learned after ten years of his disease. With no treatment or poor treatment their life expectancy will be cut by 67%. With our best present treatment there is very little increase in mortality rate. Many thousands of years of healthy human life probably are being wasted in Virginia for failure to utilize an available therapy. There are hospital beds where these diabetics can be treated and are being

treated in large numbers. There is no diabetic service where proper training and treatment are given and coordinated. Probably more hospital beds are unnecessarily wasted on diabetics than anyone realizes. Many days or even weeks are spent in the hospital today by diabetics who could have avoided this if they had known and realized the difference between following a lackadaisical treatment rather than a relatively easy but definite and continuous proper treatment.

Think what it would mean if every new diabetic went out after the initial discovery of his disease with a thorough knowledge of his problems and how to handle them! Instead of disturbing his doctor for a small boil, or a temporary digestive upset, or a bad cold, he would know how to handle himself to avoid the aggravation of the diabetes and of the other trivial condition to the point where serious illness results. He would know how to alter his diet and insulin dosage to the slight degree necessary to take care of such trivial upsets. More important still, he would recognize the symptoms that are serious, consult his doctor promptly, and thus prevent a serious or even fatal condition. The man who picked a small pimple, watched it enlarge, drain, and spread and progress to the stage of septicemia and diabetic acidosis and death could have prevented all of this easily by keeping his sugar controlled or notifying his doctor of its uncontrol in ample time to have prevented this outcome.

With the new hospitals being built and planned in Richmond and other towns in Virginia, it is appropriate that we consider a service where diabetics can be admitted and trained at the time of the discovery of their disease to avoid the pitfalls mentioned. Such a service need not be complicated nor expensive. A nurse especially trained and interested in diabetes should be in charge and she and the doctors who have patients there could give lectures and, when necessary, personal instruction to the patients each week as is done by Joslin and others throughout the country.

At the meeting of the Medical Society of Virginia at Old Point Comfort last month the Clinical Society of the Virginia Diabetes Association was formed. The aim of this group, which is composed of practitioners from various parts of the State, is to improve the diagnostic and therapeutic results in the people of this State. Any and all doctors are asked to aid in this venture in any way they wish by becoming members, by giving suggestions, and by following such procedure best suited to accomplish this goal. This seems a suitable effort in combating one of the ten major causes of death in Virginia.

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Richmond, Virginia

THE FUTURE OF MEDICINE IN VIRGINIA

M. PIERCE RUCKER, M.D.,
Richmond, Virginia.

The presidency of the Medical Society of Virginia is a great honor. Recently it has also gotten to be a big job. I am grateful for this evidence of your esteem and for the privilege of serving you at this critical time.

The November election has been variously interpreted. It has been called a mandate for compulsory health insurance. It certainly created a serious situation for medicine. Evidently, the people did not know what excellent treatment they were getting. Fortunately, however, enough Senators and Representatives did know. To quote from a speech of the Hon. Lester C. Hunt before the United States Senate, August 16, 1949, the 189,000 doctors (in the United States) are "the best trained, most skillful doctors in all the world at any time in all the world's history, doctors who have made this Nation the healthiest of all Nations. Our death rate is the lowest. Our life expectancy has risen from 35 years, when we established our Government, to 67 years—the longest life expectancy of any people on the face of the earth. Our working men lose from illness an average of only eight days a year as compared to 28 days only 49 years ago, at the turn of the Century. That is approximately one-half the days lost from work a year because of illness in England and other countries." The trouble is that the people do not know this. The Medical Society of Virginia has cooperated wholeheartedly with the American Medical Association in bringing this message to the people. It has established the most cordial relations with our representatives in Washington and it has had no small part in defeating Federal legislation that would have been detrimental to the present high standards of medical practice. The people are beginning to realize that this is not just a doctor's fight, but theirs as well. This was demonstrated by the recent decisive defeat of the President's reorganization plan No. 1. It should be remembered that Senator Byrd and Senator Robertson not only voted against this measure but *worked* against it. Had the Senate not defeated this bill, Mr. Ewing would now be a member of the Cabinet and Socialized Medicine would surely have followed.

Senator Robertson also took an active part in our Public Relations Conference in January and arranged for the meeting with our Congressmen in May. In this connection, I wish to take this opportunity to commend the work of our Director of Public Relations, Mr. Henry Johnson, who has gone about the State day and night, meeting with various lay and professional groups, explaining what the medical profession is doing for the people.

This is an opportune time to discuss the future of medicine in Virginia. Let us go back a bit and pick up the trends. When I began to study medicine fifty years ago, a revolution in medical education was beginning. The Medical College of Virginia had just adopted a four year course. As a matter of fact they were still accepting matriculations on the three year plan, provided the student had a college degree. The teaching was largely didactic. Even at Johns Hopkins, which several years previously had begun to teach medicine with emphasis on clinical instruction, the student had to get most of his clinical experience in obstetrics in the homes of the poor. There were no internships for many of the graduates. Surgery was chiefly abdominal surgery and most of that consisted of removing the appendix. "Blood work" consisted of counting the red and white cells and searching for malarial parasites in blood obtained from pricking the finger or the lobe of the ear. Rufus Cole began making blood cultures and Cooke and Boggs laid the foundation of clinical sphygmomanometry in my student days. Blood transfusions, or indeed any intravenous medication, was unknown. That hookworm existed in this country was discovered when I was a medical student but the extent of the infestation was undreamed of. Typhoid fever was a matter of course every summer and a doctor who did not have fifteen to twenty cases had no practice at all. One still heard such diagnoses as typhomalaria. Very few doctors relied on finding the plasmodium malariae to make a diagnosis. Some used the therapeutic test with quinine to differentiate malaria from typhoid fever and many

*Address of the President at the annual meeting of the Medical Society of Virginia at Chamberlin Hotel, Old Point Comfort, Fort Monroe, Va.

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could diagnose typhoid fever by the smell. The disease was thought to have a distinctive odor. Infantile diarrhea was as prevalent as typhoid fever and an infant that survived the second summer was considered to have unusual stamina. The treatment of diphtheria was on a satisfactory basis provided one made the diagnosis early. The picture of a baby almost choking to death with croup was a common one. There was no such thing as prophylaxis.

The trained nurse was even scarcer then than she is today and the doctor had to demonstrate to the mother how to give an enema or to sterilize a bottle or mix a formula. There were, of course, measles, mumps and whooping cough, but they did not make so much impression on the young doctor because their nursing care was simpler. There were no full time health officers, and the function of the health department was principally the establishment of quarantines and the fumigation of premises. About all the doctor could do with certainty was to prevent smallpox, provided he could persuade the patient to be vaccinated; cure malaria with quinine, provided he could make the patient take the medicine long enough and repeat the treatment the following spring; and cure syphilis with mercury and iodides if he could control the patient for two years. He did not have a laboratory diagnosis for syphilis but had to rely on a clinical diagnosis. The surgeon could cure appendicitis. There was no orthopedic surgery to speak of. X-rays were used only to diagnose broken bones, and there was no radium. Life expectancy was less than 40 years.

Now the medical student gets four years of laboratory and clinical training in the medical school and spends from one to six years in a hospital. The Health Department, which was then practically non-existent, now has a health officer and a team of health nurses in almost every county. The State Health Department now spends nearly three million dollars protecting our health. Then, the citizens of Richmond drank James River water raw. Now, the mud and the bacteria are removed and the Ph is kept constant at 8.4. Milk is inspected from the dairy farm to the delivery wagon. It is pasteurized and safe (and expensive). Typhoid fever has disappeared and the babies go through their second summers without mishap. Surgery of the brain and of the chest is as common as was abdominal surgery fifty years ago. The blood is subjected to all sorts of chemical and serological tests. Blood transfusions and intravenous therapy are so common that one marvels that the young doctor can find so many superficial veins. Hookworm disease has been eradicated for so long that it has been forgotten. A single case of typhoid fever makes the front page of the newspapers. The doctor of today has specific cures for most of the infectious diseases and a six day cure for syphilis and a single shot cure for gonorrhoea. There are efficient vaccines and inoculations against many of the infectious diseases. With the exception of poliomyelitis, the epidemic diseases have been conquered. Life expectancy is now more than 67 years.

Tuberculosis is no longer first among the causes of death, but is a poor seventh on the list. The past few decades have been an era of specialization. This has undoubtedly led to a tremendous increase in knowledge, but it has also increased the cost of medical care. The public had begun to think that only the specialist could properly care for the sick. The pendulum has already begun to swing back to a more sensible opinion. The family physician, or general practitioner, has an important place in medicine and can take care of most situations demanding medical care better than the specialist and at less cost.

Specialization has taken a new turn. Each major disease has an association or foundation back of it that is busy with the problems of that particular disease. These are composed of medical as well as lay persons. The oldest of such groups is the American Anti-Tuberculosis Society which blazed the way in education of the public in health matters. The American Cancer Society is engaged in a nation wide campaign for routine health examinations as a means of detecting cancer early when it is amenable to treatment. It encourages research and provides facilities for diagnosis and treatment for those who need it. The American Heart Association has done valuable work in standardizing the classification of that disease. The group that is interested in rheumatic fever is developing the care of these children as a community project, with special care of the disease in foster homes and provision for schooling at the bedside and manual training adapted to the patient's physical needs. The aim is to protect these children from recurrences and at the same time help them to find a useful place in society. Everyone knows about the

Infantile Paralysis Foundation and its annual mile of dimes. The Chronic Arthritis Society is less well known. Just recently the Society for the Study of Diabetes has sponsored a Diabetes Week with emphasis on early diagnosis. There are a million unrecognized diabetics in the United States and Canada and statistics show that complications in the unknown diabetic are fifteen times more fatal than complications in the recognized diabetic.

The newest of all medical societies, The American Academy of General Practice, introduces a new principle, i.e., membership for a limited period of time and renewable only upon continued postgraduate study. The certificate is granted for three years. At the end of each three year period the fellow must produce evidence of having taken 150 hours of satisfactory postgraduate work or the certificate will not be renewed.

Thus medicine faces the future with better trained personnel than at any time in its history and is taking steps to insure its continued improvement. On the other hand, it faces more and different problems. The population is becoming decidedly older. It is estimated that by 1960, 9.1 per cent of the population will be 65 years old or older. A new branch of medicine, geriatrics, has arisen. Old folks present entirely different problems, but the problems are not insurmountable. With anesthesia developed especially for the aged, operations are being performed successfully that were not thought possible ten years ago. More study must be given to developing suitable and convenient facilities for social, recreational and cultural activities that are within the physical capacity of older people. If we are to have old age we must make it endurable.

A glance at the ten leading causes of death in 1900 and in 1946 shows where the future problems lie. In 1900 tuberculosis had a death rate of 195 per 100,000 population, pneumonia a rate of 176, diarrhea and enteritis a rate of 140, diseases of the heart 137, nephritis 89, accidents and violence 88, cerebral hemorrhage 77, cancer 64, bronchitis 45 and diphtheria 40. In 1946, heart disease was the leading cause of death with a rate of 307, cancer was second with a rate of 130, accidents and violence had a rate of 88, cerebral hemorrhage 78, nephritis 58, pneumonia 38, tuberculosis 36, premature birth 29, diabetes 25, and arteriosclerosis 17.

In 1900 the three leading causes of death were bacterial diseases and five of the ten leading causes of death belonged in that class. In 1946 only two bacterial diseases were in the ten leading causes of death and they ranked sixth and seventh on the list.

While bacterial diseases have been practically conquered, the virus and Rickettsia diseases present an almost unexplored field. We know the size and shape of many of these ultra microscopical bodies but nothing of their structure. Many have extremely long incubation periods, notably hydrophobia and infectious hepatitis. Some are transmitted by inadequately sterilized syringes, some by the bite of animals, some by insects and many in ways that are as yet unknown. Rocky Mountain spotted fever, a disease that was believed to be confined to certain valleys of the Rocky Mountains, is now found to be more prevalent in Virginia than anywhere else in the United States. Fortunately, we now have two specific cures for it and, in addition, means of inducing a short lived immunity. The most encouraging feature of these diseases is that many of them confer a lasting immunity. Some of the most brilliant achievements in medicine have been in connection with these diseases, even long before there was any knowledge of viruses. Jenner's experiments with cowpox, Pasteur's work with rabies, and Walter Reed's work with yellow fever are unforgettable examples.

More and more of the doctor's time must be directed to the degenerative diseases. The obstetrician has a problem in the premature infant. Substantial progress is being made with two of the commoner causes of prematurity, i.e., toxemia of pregnancy and placenta previa. With the more general acceptance of prenatal care, serious cases of toxemia of pregnancy are being prevented. More recently, new ideas in the treatment of placenta previa have been advanced. It used to be taught that a patient with placenta previa should be delivered as soon as the diagnosis was made. The result was a high fetal mortality on account of prematurity. Now some clinics are advocating letting these patients alone until they reach term and when such a course has been followed, there has been no increase in the maternal mortality, and a reduction in the fetal mortality from 30 or more per cent to 6 per cent. Newer work with endocrine therapy indicates that premature births from other causes may be prevented.

The pediatrician of the future will practice

XUM

chiefly immunology and dietetics. Already he has practically eliminated diphtheria, scarlet fever, and whooping cough from his practice and, before he turns his patients over to the other practitioners, he will have immunized them against tetanus and typhoid fever. When the public forgets these diseases, he will then have a major task of continuous education or the public will become careless. Just recently, for instance, in England they have repealed the compulsory vaccination law.

As diseases become more chronic and as the population becomes more industrialized, our diagnoses must be keyed to early recognition of the preclinical phases of disease and our treatment to rehabilitation. We must become health conscious. Our examinations and laboratory tests must be directed more and more to the study of the reserves of the various organs. We will not be so much interested in heart murmurs, arrhythmias etc., as in how much margin of safety a given heart has. Such tests as the sugar tolerance test will be more in use than the test for the presence of glucose in the urine. The mother who has an over-sized baby will be warned of the possibility of having diabetes in the next four or five years, and she will be advised to avoid weight gain herself. Our therapy must become preventive on the one hand, and on the other hand, it must be extended to include rehabilitation. If a patient loses an arm, a leg or an eye in an accident, it is recognized that the job is only half done when he is cured of the immediate effects of the accident. He must be restored to a useful place in society where he can be happy. Perhaps he will need some plastic surgery and perhaps he will have to be taught a new trade or handicraft. He will need a social worker and an industrial instructor or a psychologist in addition to his strictly medical or surgical attendants. This need is already recognized in case of accidents. The same position is being taken in regard to chronic and crippling diseases. A child with rheumatic fever has a poor outlook if treated only medically, but if a team of social workers, educators etc., take charge of him, and teach him how to take care of himself and to make a living by some means within the capacity of his heart, he can become a useful citizen. The hypertrophic arthritis patient will need a lot of physiotherapy with his ordinary therapy, but his educational needs will not be so great as those of the rheumatic fever patient. It seems quite likely that the availability of new drugs will soon make the last sentence obsolete. The cured tuberculosis patient needs placement advice and supervision or his cure may not last. The patient with the type of heart disease that now kills most of our people needs vocational and recreational advice as much as, or even more than, medical advice. The cure of cancer is probably right around the corner, but until it is discovered much can be done to reduce its ravages by early diagnosis and treatment. The only way to diagnose cancer in its early and curative stages is to *look* for it and this should be done systematically and periodically.

Finally, with a constantly aging population with nearly ten per cent at the retiring age, the doctor must be an expert on hobbies. Many men and women have gone through life without learning how to play. When they are no longer able to work they have nothing to interest them. The doctor must be able to fit them with a suitable hobby.

What about atomic energy? Much has been said about the destructive effects of this new power. At first we were told that everyone in and about Hiroshima and Nagasaki was killed. Later we were told so much nuclear radiation is released when a bomb explodes that it is unsafe for rescue workers to go into the bombed areas. The very water and soil is activated and plants and animals growing in such environs can concentrate many radioisotopes with long half life. Now it seems that more than 200,000 of Hiroshima's 300,000 inhabitants survived. Thousands who did not succumb immediately to blast and thermal injury presented new and bizarre types of radiation sickness. Some were deprived completely of hair, some developed mahogany pigmentation and others were depigmented. Some developed intractable intestinal trouble, and others had all of their bone marrow destroyed and died as soon as they used up all the blood cells they already had, a matter of a few weeks. The full effect on the gonads can not be determined for several generations.

There is one comforting thought about atomic energy, and that is that man has a healthy respect for it and so far has devised means of handling it with safety. Until recently there had been no report of anyone working at Oak Ridge or other experimental laboratories having been injured. This is in marked contrast to the early experience with Roentgen rays. Many of the earlier

XUM

experimenters with x-rays lost fingers, ears or developed incurable ulcers. Some even lost their lives. Only trained workers are allowed to handle the atomic material.

With the great variety of ionized atoms, the possibility for study is unbelievable. As yet, medicine has used it only in investigation. An atom such as sodium or iodine with a short half life is ionized and such tagged atoms are traced in the various tissues of the body. There has been some experimentation with cancer, but nothing better than radium or x-ray has been found.

The doctor of the future will be less of an individualist than he has been in the past. More people are interested and anxious to help in his work than ever before. He will have more and probably adequate facilities and nursing help for his work. While his problems are becoming more complex, he will have more skilled help and more complicated machinery with which to solve them. Financial aid for research problems is becoming more readily available. Even his therapy is becoming a matter of team work. The parson and the priest have always been available in helping the doctor solve family problems. Now he has the social worker, the marriage counsel, or the vocational planner and other personnel of the Educational Department, the physiotherapist and the psychologist.

The accepted technic of good prophecy is to lay down bounds within which the prophecy will act. If such qualifications are not met, the prophecy is off. The storm now passing over the Rocky Mountains will bring snow to the Atlantic Seaboard provided a high pressure area does not develop over the Gulf of Mexico. For medicine the big "if" is Federal control. In no country has bureaucratic medicine prospered. When it became firmly established in Germany, the quality of medicine declined so markedly that Germany lost her preeminence in medicine. When I was there in 1905 the beginning of the decline was noticeable. There was a brutality in the treatment of patients which I, in my ignorance, attributed to the docility of the German peasant and the overbearing nature of the German ruling classes. I still have a mental picture of eight or ten patients in the Leipzig clinic who had been sitting on a hard bench with heads bent back and to the right, so as to accommodate a rigid tube in the esophagus. The esophagoscopes had been introduced by an assistant and the patients were waiting for the Herr Professor to glance down their gullets—the quintessence of bureaucratic medicine.

The same system was put into operation in England on July 5, 1948. Already obstetric and dental anesthesia and endocrine therapy have been banned because the authorities consider them to be unnecessary. By the end of the year the costs had so far exceeded the estimated costs that editorials in the newspapers were discussing the need of devaluating the pound sterling.

In the past, Virginians like William Baynham, Jesse Bennett, Ephraim McDowell, John Peter Mettauer, Thomas A. Emmett, Lewis G. Pedigo, Hugh Cumming, Henry Rose Carter and Walter Reed have made worthwhile and even epoch-making advances in medicine and surgery. The future holds bright promises for continuing such work in ever larger fields—if the Congress does not put the blight of Federalized medicine upon us.

Drugs Cut Death Rate.

Modern methods of treatment have reduced the mortality rate of myasthenia gravis to about 10 per cent, according to two doctors from the University of Texas School of Medicine, Galveston. In this rare disease, the cause of which baffles doctors, the victim's muscles progressively become weaker. The muscles most frequently affected are those concerned with movements near the eyes, with resulting squinting and "seeing double." Generalized muscular weakness also occurs. Untreated, the disease runs a fatal course in 50 to 75 per cent of cases in a few years, Drs. Charles T. Stone and J. Alfred Rider write in the September 10 *Journal of the American Medical Association.*

Drugs, principally neostigmine and tetraethyl pyrophosphate, give complete relief in some cases and have greatly reduced the mortality rate of the disease, which is now probably about 10 per cent.

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THE ADVANTAGES AND LIMITATIONS OF HORMONE THERAPY IN BENIGN AND MALIGNANT PROSTATISM*

LINWOOD D. KEYSER, M.D.,
Roanoke, Virginia.

While the prostate itself elaborates no specific hormone, it is the object of an interplay of endocrine influences which have a far-reaching significance. The testes, the pituitary and adrenals affect especially the development, growth and size of the prostate gland. Also they seem to have a definite but not too clearly defined influence on its regressive changes in senescence, its hyperplastic manifestations and to modify the growth and development of its neoplastic diseases.

THE TESTICULAR HORMONE

Following McGhee's isolation of an active lipoid soluble male sex hormone from the bull's testes in 1927, Butenandt, Ruzicka and others isolated androsterone, dehydroandrosterone and testosterone. Subsequently the relation of these substances to cholesterol and their synthesis from this chemical was established. It is now believed that testosterone is the true male hormone, androsterone and dehydroandrosterone being less active metabolic derivatives found in urine.

It had been known that castration in lower animals was followed by atrophy of the prostate and seminal vesicles. When androsterone was isolated, it was found by biologic assay that virility, sex vigor and the occurrence of prostatic hypertrophy in later life appeared concomitantly with a diminution of androsterone in the urine. Moore and Miller noted an especial decrease in older men with prostatic hypertrophy. Hence the initial but somewhat erroneous assumption was made that these phenomena of senescence were due to a decrease in male sex hormone function.

THE PITUITARY HORMONE

Hoskins in 1911 had developed the concept that the gonadotrophic activity of the pituitary was normally held in check by the gonads. Smith and Engle showed that hypophysectomy in animals caused atrophy of the prostate, and Walsh, Cuyler and McCullagh found that such post-hypophysectomy prostatic atrophy in rats could be prevented by daily administration of male sex hormones. Also it was noted by Korenchevsky, Nelson, Gallagher and others that castration was followed by alteration of secondary sex characteristics, hypertrophy of the pituitary with the formation of peculiar vacuolated cells in the basophilic elements (the so-called signet cells or castration cells) and hypertrophy of the adrenals. Such changes could be prevented by administration of male hormone in large amounts or of lesser amounts of male hormone and extremely small amounts of estrogen.

From female endocrinology it had been established that there are two gonadotrophic hormones in the anterior pituitary gland, one producing follicle stimulation, the other producing luteinization. In the male evidence is accumulating to show that the follicle stimulating hormone activates the seminiferous tubules to produce sperm, while the luteinizing pituitary factor stimulates the interstitial cells of Leydig to elaborate the androgenic hormone, which in turn stimulates the development of secondary sex characteristics.

Following the work of Meyers, Vidgoff and Hunter who somewhat paradoxically produced atrophy of the prostate by feeding rats with desiccated beef testes, Lower and McCullagh, from experimental evidence, developed the concept that the testis has two major hormones. One, "inhibin", a water soluble hormone, developed in the tubules or the germinal cells, inhibits or depresses the gonadotrophic activity of the pituitary from over-stimulating the interstitial cells of the testis. The other, "androitin", is produced by the interstitial cells of the testis. Androitin closely related to androsterone and testosterone acts as a sex stimulant and in excess produces prostatic hypertrophy. The Lower theory simply stated is that after middle age regressive changes in the germinal cells of the testis lessen the elaboration of the pituitary depressing inhibin. The over-active pituitary then stimulates an over-production of so-called androitin (male hormone) with consequent prostatic hypertrophy. It had been noted before that parenteral injection of the anterior-pituitary-like principle of pregnancy urine would produce prostatic enlargement in cas-

*Presented before the annual meeting of the Medical Society of Virginia in Richmond, October 18-20, 1948.

trated rats and monkeys. Whether or not this enlargement is similar to prostatic hypertrophy in man is a matter of dispute. However, the Lower theory has not been accepted. There is neither enlargement of the pituitary in benign prostatic hypertrophy in man nor are histologic changes noted; there is no prostatic hypertrophy in teratomas of the testis where large amounts of circulating substances similar to pituitary gonadotrophic hormone are present; there is not a consistent finding of increased urinary excretion of significant amounts of either gonadotrophic substances, male hormone, or estrogen in patients with prostatic hypertrophy.

Estrogen or female sex hormone now enters the picture. Zuckerman showed that male and estrogenic hormone are both elaborated in the male organism. He felt that they are balanced in such proportion that the estrogenic powers of the female sex hormone are ordinarily inhibited. Senescence disturbs this balance. Wugmeister in 1937 advanced the idea that the female hormone diminishes in later life in both male and female whose organisms tend toward "hypervirilation". The anterior pituitary, being no longer inhibited by estrogen, overstimulates the testis to elaborate an excess of androgen and thus stimulates the genital apparatus to hypertrophy.

Further concepts of estrogen-androgen imbalance as a cause of prostatic and genital changes were numerous. Zuckerman and Parkes produced prostatic enlargement in castrate monkeys by estrone injection and counteracted it with injections of testosterone. The enlargement is due to an increase in the fibromuscular elements, with hyperplasia and metaplasia of the ejaculatory ducts, prostatic collecting tubules, the epithelium of the urethra and vagina masculina. The epithelium of the true prostatic glands did not change. Geschickter by injecting testosterone restored the prostatic epithelium and produced hypertrophy of the seminal vesicles in monkeys whose prostates had undergone atrophy from castration. Lacassagne, Burrows and others demonstrated that administration of estrogens to mice produced metaplasia of the prostatic epithelium (a common finding in prostatic hypertrophy). Von Cappellan, Burrows and Zuckerman concluded that large amounts of estrogen acting during presenility would cause enlargement of the prostate. If true, large doses of androgen should prevent and relieve prostatic hypertrophy. However, Moore, McClellan and others found that injection of testosterone in large doses resulted in no significant restoration of the involuted prostate of presenility or in the histologic appearance of the benign hypertrophied prostate. While injection of estradiol benzoate produces conspicuous alteration in the urethral and ductal epithelium, there is little if any change in the tissues of benign hypertrophy.

However, Huggins has demonstrated that injection of testosterone restored not only the size but the secretory activity of atrophied prostates of dogs following castration.

Again showing how conflicting the evidence may be when different workers deal with different species under varying conditions, we may cite Korenchevsky and Dennison who produced prostatic atrophy in normal rats with estrone and prostatic hypertrophy in castrated rats with the same female hormone; Deming and coworkers' demonstration of prostatic hyperplasia in a male macaque monkey with estrone; a converse observation of hypertrophy of the rudimentary prostatic analogue in female mice with testosterone but not with estrone. This evidence supports the view that over-balance of female hormone in males and of male hormone in females might produce hypertrophy of the prostate or of its contra-sexual homologue in certain animals. Wattenberg and Moore note that the utricle and prostatic urethra are homologous to the female vagina. They found that the clinical use of estrogens produced aquamous hyperplasia of these structures, such as have been noted experimentally in mice and monkeys.

In general, the experimental evidence of hormonal relationship to prostatic enlargement is confusing and highly controversial. It has been of value, however, in suggesting certain avenues of approach to the clinical use of hormones in therapy.

THE NATURE OF PROSTATIC ENLARGEMENT

Deming and his coworkers from histologic studies contend that the components of the prostate gland responsible for benign enlargement are the muscle fibres (originally from the lower Mullerian tube) and the prostatic ducts. The process begins as a solid fibromuscular mass of Mullerian tissue in the muscular wall of the prostatic urethra. This mass stimulates an epithelial proliferation of the ducts. The primary tumor is then invaded by the duct epithelium which forms glandular tissue and grows

XUM

more rapidly than the stroma. Hence the mass may appear wholly glandular. The glands of Albarran and the glands of the posterior and lateral lobes are not involved in the early phases of development. Thus benign enlargement is a hyperplasia and not a hypertrophy of tissue. They compare this with the growth of uterine myomata and note that hypothetically they have a common anlage. The hyperplastic growth causes compression atrophy of normal prostatic tissue which is displaced laterally by the growing adenomata. These authors contend that hormone therapy does not affect the hyperplastic tissue and does not cause regressive changes in the normal prostatic tissue which is already atrophied by compression.

THE INCIDENCE OF BENIGN AND MALIGNANT PROSTATISM

In 35% to 50% of men over 50 years some hyperplasia of the prostate is noted. A variable number—from 20% to 30% of these—will require treatment for obstruction. The incidence of cancer as a histologic finding is impressive. In 1935 Rich noted 41 cases of prostatic cancer in 252 autopsies in men over 50 years (16%). Moore in 1935 observed cancer in 262 prostates from patients; none under 44 years; 17% from 41-50 yrs.; 14% from 51-60 yrs.; 23% from 61-70 yrs.; 21% from 71-80 yrs.; and 29% from 81-90 yrs. Baron and Angrist found histologic evidence of cancer (occult adenocarcinoma) in 46% of 50 prostates studied by serial sections. The prostates were from unselected consecutive autopsies in men over 50 yrs. Metastases were not noted. In general the incidence of hypertrophy is three times that of cancer.

Carcinoma of the prostate is frequently associated with benign hypertrophy but probably arises independently of it. In about 75% of the cases the disease begins in the posterior lobe which is almost never the seat of benign hyperplasia. Adenocarcinoma, medullary, scirrhus and even squamous celled varieties are found and may frequently be associated in the same gland. The growth of prostatic cancer is insidious and variable. Certainly we do not see carcinoma of the prostate clinically in the same degree of frequency that is noted histologically at autopsy or especially in serial sections of glands removed at autopsy. Sarcomas of the prostate are exceptionally rare. While they may occur at any age, most cases are encountered in youth.

CLINICAL USE OF HORMONE THERAPY IN BENIGN PROSTATISM

Male hormone therapy, chiefly testosterone, has been widely used for benign prostatic hypertrophy. Walther and Willoughby reported 15 cases so treated with temporary clinical improvement. Day, Cary, Boland and others reported similar cases which responded clinically to testosterone but no changes in size were noted by cystoscopy or by rectal palpation. Heckel was not impressed with the use of testosterone in prostatic hypertrophy but did find that oligospermia resulted from continued large doses.

On the whole the use of the male hormone has not been satisfactory. It may act at times by inhibiting the pituitary or by stimulating the musculature of the bladder (Kearns) to greater propulsive activity or perhaps by exciting indirectly the adrenal cortex to activate steroidal hormone influence on the prostate. It may also supply an androgenic deficiency, counterbalance an estrogenic excess, thus restoring a theoretical androgen-estrogen imbalance. *It must be remembered in considering the effects of all hormone therapy that benign low grade prostatic obstruction normally is associated with periods of regression.* Some authors find that the effects of testosterone therapy are no better than those achieved in a parallel series of cases when injections of saline are given (Draper). Certainly no specific changes in size of the gland or regressive histologic changes in its tissues by biopsy have been noted.

The use of estrogens in treating benign hypertrophy, however, cannot be dismissed so quickly. With Huggins' introduction of antiandrogenic therapy in prostatic cancer there also arose much popular interest in the hormone therapy of benign hypertrophy. Huggins, Stevens and Hodges subjected 3 patients with benign hypertrophy to castration. This was followed by epithelial atrophy together with gross decrease in the size of the gland as revealed clinically and by biopsy specimens. They felt that as old age approaches there is a decrease in the male hormone, yet enough androgen remains to produce prostatic hypertrophy. However, if this residuum of hormone is eliminated or reduced by castration the prostate will atrophy. Also by the

administration of estrin without castration the remaining male hormone is neutralized and atrophy similar to that produced by castration takes place.

Estrogens, chiefly diethylstilbestrol, have been widely used during the past ten years. Kahle and Maltry, Kittridge, Klein and Newman have noted a fairly high degree of clinical improvement in a variable percentage of cases thus treated. In some instances the size of the prostate noted by cystoscopy and rectal examination was definitely diminished and biopsies showed regressive changes in the prostatic tissue. On the other hand, Pierson, by roentgenographic measurements of the prostate in 13 patients with retention of not over 3 ozs., noted a variable diminution in the size of the prostate averaging about 7 mm. in 10 cases after treatment with daily small doses of stilbestrol. He felt that the shrinkage was not great enough to warrant this therapy except in unusual cases.

Since 1941 I have applied both estrogenic and androgenic hormone treatment to 110 patients with benign prostatism in various degrees. Their ages have varied from 50 to 87 yrs. Sixty-two of these had little residual (none to 3 oz.) but complained of weakening of the stream, frequency, nocturia and dribbling. A variable degree of enlargement by rectum was noted. (It is the prostatic tissue growing into the bladder neck and not that felt bulging into the rectum which gives obstructive symptoms.) In short, the majority of these were borderline prostatics and usually younger men. Estrogen therapy gave clinical relief to about two-thirds of this group of 62. The treatment was frequently combined with massage, diathermia and dilatation of the urethra with sounds. In approximately one-half of the remainder we were not impressed with the treatment but operation was considered unnecessary or refused. The remainder developed sufficient obstructive symptoms to warrant operation at my hands or elsewhere. When estrogen therapy gave favorable response the glands became softer, seemed to be reduced in size and the stream became stronger. While taking the matter of spontaneous remission into consideration, it was felt that in this group of cases estrogen therapy is especially effective in a considerable proportion. In short, the younger the patient, the softer the consistency of the gland, the more likely the relief from estrogen. Hard fibrotic glands and those in arteriosclerotic mentally confused older men respond to such treatment less strikingly and require frequent passage of sounds or even surgery if there is a residual urine of over 3 oz.

The remaining 58 patients were prostatics in advanced stages of obstruction, i.e., with a residual of 3 oz. or more. The results of estrogen therapy, while brilliant and spectacular at times, have not been consistent. In the 58 cases of this type estrogen therapy reduced residual, the blood urea level if elevated was diminished, the stream enlarged and operation has been declined in 38. Some of these patients improved at first but later developed sufficient symptoms and clinical findings to warrant surgery. Nine patients, because of senility, uremia, concomitant fractures or surgical procedures needed at other sites, were subjected to suprapubic cystostomy. After estrogen therapy their sinuses closed and remained closed, the patients voiding well and with little or no residual urine. Another group of patients (about 20 in number) was placed on indwelling urethral catheter drainage and, after estrogen therapy, had such reduction of residual urine, increased force of stream and general well-being that operation was refused. Several of these returned later for operation and others should undoubtedly have done so.

Diethylstilbestrol, a synthetic chemical having estrogenic action, in doses varying from 1 mg. to 20 mg. daily given by mouth or injection has been the drug most used. The dosage is not standardized in the treatment of either benign or malignant prostatism. With the optimum dosage unknown, most authorities fell that larger doses are unnecessary. I usually start with 5 mg. daily and increase or decrease the dose to the patient's tolerance.

The drug has certain hazards, viz., nausea (a very frequent finding), dizziness, edema of the lower extremities, and an erythematous skin rash, at times leading to exfoliative dermatitis. Loss of libido and oligospermia result from large doses and must be considered in treating younger men or older ones who are sexually active. Painful engorged breasts occur in a large proportion of cases on prolonged therapy or with larger dosage. Stopping stilbestrol and administration of testosterone apparently helps to relieve this breast engorgement. However, testosterone does not entirely neutralize estrogen effect nor has the exact quantity of the dosage needed to depress estrogen action been determined. In a small group of cases I have used oreton (tes-

XUM

tosterone propionate) alternately with courses of estrogen and at times apparently with good result.

Premarin, estradiol-benzoate and estinyl have been used with variable result when diethylstilbestrol has not been tolerated. It is possible that various estrogens act differently in different individuals according to their individual liver metabolism.

In summary, estrogen or androgen therapy is no substitute for surgery in patients with well developed prostatic obstruction. *The accurate evaluation of hormone therapy becomes most difficult when we remember that prostatism frequently regresses and symptoms subside for long periods of time when local measures such as massage, diathermia, drainage or even no therapy is applied.* Nevertheless, hormone treatment, especially with estrogens, appears to be of value in borderline cases with small residual and soft glands. It may be tried with occasional success in larger obstructive glands. If after one to two weeks treatment no sharply defined and obvious improvement is obtained, resection or prostatectomy is in order.

Recently I have been preparing certain patients with large glands for prostatectomy by a preliminary course of estrogen, if the delay is warranted. At operation the gland has seemed smaller, firmer and perhaps less vascular but I cannot yet speak conclusively on this point.

Certainly the urologist should explain to his patient that estrogen therapy is a trial that, should he get an immediate favorable clinical response, frequent observation with periodic courses of the drug will probably be needed. If the patient's response is not immediately definite and there is no contraindication, he should be submitted to surgery.

ENDOCRINE THERAPY IN PROSTATIC CANCER

The application of endocrine therapy by Huggins and Herbst to the treatment of prostatic cancer is one of the outstanding contributions to modern science. It marked the first signal response of malignant disease to chemotherapy. We may state the concepts developed by this work as follows. The histogenesis of prostatic carcinoma is not altogether autonomous. These neoplastic cells are stimulated by androgens from the testis and to a lesser degree from the adrenals. The effort to eliminate or reduce to a mininum this androgen stimulation has been carried out (1) by surgical castration, (2) by castration with x-ray irradiation, (3) by administration of estrogens with or without castration to neutralize or depress androgens, (4) by adrenalectomy or adrenal irradiation to eliminate a secondary source of androgen formation after castration and (5) by pituitary irradiation as a means of suppressing pituitary stimulation of androgen elaboration.

Prostatic cancer cells freed from androgen stimulation undergo retrogressive change. Decrease in size of nuclei, condensation of nuclear chromatin, absence of nucleoli, clearing of the cytoplasm, rupturing of cell membranes with coalescence of vacuoles, with ultimate clustering of pyknotic nuclei and fragments of cells in the center of acinar spaces have been noted (Kahle, Shenken and Burns). All the cells do not die but after a variable period of time they resume a recurrent but frequently decreased activity.

Estrogen inhibits pituitary activity, depresses the germinal epithelium of the testis and, to a variable degree, neutralizes the male hormone or diminishes its elaboration. Excessive estrogen administration may produce carcinoma of the breast in male mice. The possibility of such a result in the human male and female has been suggested.

SIGNIFICANCE OF SERUM PHOSPHATASE

Huggins found that the serum acid phosphatase was usually elevated above the normal (3 to 5 King Armstrong units) in advanced prostatic cancer, especially if bone metastasis is present. The neoplastic epithelium itself showed large amounts of this enzyme as determined by specific staining methods. Acid phosphatase, present in traces in the prostate of infants, is found in large amounts in normal adult and hypertrophic glands. It is even more abundant in prostatic cancer cells. Grollman holds that acid phosphatase in prostatic cancer does not enter the circulation in detectable amounts until the neoplasm breaks through the capsule with local infiltration or distant metastasis. Then the serum acid phosphatase becomes elevated in as high as 85% of patients (Sullivan, Gutman and Gutman). In a fair number of patients, however, with extensively disseminated lesions this enzyme remains normal in the blood (Alyea and Nesbit). Febrile reactions may temporarily reduce elevated serum acid phosphatase even in advanced cancer.

Castration is usually followed by a sharp drop of the elevated serum acid phosphatase within a

week. This occurs more slowly (in 2 to 3 weeks) after estrogen administration. Failure of the elevated serum acid phosphatase to fall after castration or estrogen administration is usually accompanied by no clinical improvement. Furthermore, it is a sign that the patient will not respond to antiandrogenic therapy and that the cancer cells have become autonomous and are no longer androgen dependent.

Serum alkaline phosphatase is frequently elevated above normal (6 to 10 King Armstrong units) in prostatic cancer. It reflects activity of bone around tumor metastasis. Patients with bone metastasis have an increase in level of serum alkaline phosphatase in about one-third of the cases. In a large number following castration there is a sharp rise followed by a gradual decline in the level of this enzyme to normal, reflecting a decreased metastatic activity with bone healing. The serum alkaline phosphatase is less likely to rise in patients treated with estrogens.

Elevated serum acid phosphatase has come to be regarded as evidence of bone metastasis and its rise or fall is interpreted by many authors as an index of response to endocrine treatment. Its rapid or progressive rise is an unfavorable sign. Huggins felt that failure of the serum acid phosphatase level to fall after orchiectomy may be due to extratesticular androgen formation probably in the adrenal cortex and that this feature was an indication for estrogen therapy. In many of these cases it is also probable that the tumor cells have become androgen independent. Contrary to these views, Nesbit feels that the serum acid phosphatase is not a reliable index of metastatic activity.

SEDIMENTATION RATE OF ERYTHROCYTES

Numerous authors have called attention to the frequent finding of accelerated erythrocyte sedimentation rate in malignant disease especially when metastatic. Boylan and Tillisch and others have studied the matter in relation to prostatic cancer and feel that the sedimentation rate should be taken into consideration with acid phosphatase levels in evaluating the activity of this disease.

THE SIGNIFICANCE OF THE 17 KETOSTEROIDS

These substances are steroids having a ketone group at the 17th carbon atom. They are produced in the testis and adrenal cortex and their quantitative estimation in the urine is regarded as an index of the androgenic activity of these organs. They include androsterone and related compounds as well as estrone. The estrone can be eliminated by treatment with alkali leaving the urinary androgens which can be further divided into alpha and beta fractions, the latter being precipitated by digitonin.

Several authors have studied the excretion of 17 ketosteroids in prostatic disease, but Frame and Jewett, and Satterthwaite, Chute and others found that there were insignificant differences in the daily excretion of these steroids in the urine of prostatic cancer patients and in that of normal men in the same age group. Huggins and Scott found in four cases of prostatic cancer treated by adrenalectomy after castration as a further antiandrogenic regimen that the 17 ketosteroids practically disappeared.

The general opinion is held at present that 17 ketosteroid determination is not of great prognostic value in treating prostatic disease. Castration decreases estrogen urinary excretion and some authors state that it causes a late rise in urinary androgens (17 ketosteroids) from adrenal activity. Likewise, the pituitary gonadotrophic hormone excretion in the urine rises following castration. Estrogen (stilbestrol) administration decreases 17 ketosteroid output and that of the pituitary gonadotrophic hormone. However, estrogen administration causes a rise in urinary estrogen assays.

CLINICAL TREATMENT OF PROSTATIC CANCER

Antiandrogenic therapy of prostatic carcinoma centers chiefly around castration and the administration of estrogens. Castration in a majority of cases is followed by relief of pain, increase in appetite, in hemoglobin and in general well being. However, there is loss of libido and the power of erection. The prostate becomes softer and diminishes in size. Bony metastases frequently show increased calcification by x-ray and at times apparent healing. Frequently patients have hot flashes which can be controlled by estrogens. Injection of testosterone probably reactivates the growth as pain becomes intensified and the serum acid phosphatase level rises. In performing orchiectomy only the glandular tissue of the testis is removed but this must be done thoroughly.

Estrogen therapy if successful produces these results more slowly but some outstanding urologists such as Kearns (who uses estinyl 0.05 mg. daily) and Dean advocate estrogen treatment ini-

XUM

tially. Herger and Sauer, and Nesbit ran parallel series of cases, one treated initially by castration and the other initially by stilbestrol. Herger and Sauer felt that estrogen is useful initially in selected cases but that these should have frequent examinations and that castration should be done when changes in prostatic size, infiltration of the pelvis, obstructive symptoms or elevation of the serum acid phosphatase are noted. Nesbit's clinical results from castration and estrogen therapy were somewhat identical but there was more regression in the size of the gland in patients treated with castration. Pain in certain patients after castration was relieved by stilbestrol. Herbst, Emmett and Greene, and Alyea advocate, as a rule, initial castration followed by estrogen in varying dosage and type. Deming feels that castration and estrogen produce about the same result. In general it is agreed that castration should be the initial procedure when extensive malignancy and metastasis are present and when the patient does not show a favorable response to estrogen. However, when castration has not produced a favorable response, as a rule estrogen will not bring improvement. Alyea, in a series of 40 patients treated by orchiectomy and followed over a 3 year period, found that 32% were dead in 2 years. Of 23 with metastasis, 32% were dead within one year and 42% within 2 years.

Munger has been the champion of x-ray irradiation of the testis as a means of castration. This is combined with regional irradiation. Others have suggested irradiation of the adrenals and even of the pituitary, especially when reactivation of the cancer occurs following castration and estrogen therapy. However, such measures have not attained wide acceptance.

The life expectancy following antiandrogenic therapy has naturally not been consistent in the numerous reports such as those of Alyea, Birdsall, Wear, Carroll, Seamans and others. Bumpus in 1926 found that in 485 cases when no treatment was given the average duration of the disease from the first symptoms to death was 31 months. Surgery and irradiation prolonged life but little, while relief of obstruction (at that time by cystostomy) on an average prolonged life to 57 months. The variability of cancer in its grade and progress in the individual patient, the extent of the lesion at the time treatment is undertaken and other factors make it difficult to answer the question as to how much longer patients will live after antiandrogenic treatment than if they had not been given this therapy.

Antiandrogen Treatment Preliminary to Total Prostatectomy

Recently certain urologists, among others Colston and Gutierrez, have proposed castration and estrogen treatment in selected cases of prostatic cancer which have progressed just beyond the stage where radical total prostatectomy is indicated. When the gland responds by shrinkage and periprostatic induration subsides, total prostatectomy may be performed with operative recovery as a rule. This use of antiandrogen therapy is hopeful but we must await the passage of time to evaluate its efficacy.

Discussion

While it is difficult to crystallize a definite program for treating prostatic cancer, certain tentative conclusions may be drawn:

1. When the growth is confined within the prostatic capsule, the serum acid phosphatase level normal, and metastases are absent, it is generally agreed that radical total prostatectomy (preferably after biopsy) together with removal of the seminal vesicles, offers the one hope for permanent cure.
2. Antiandrogenic therapy by castration and estrogen administration will cause regression of the growth for a variable period of time during which period the patient will frequently be relieved of pain and enjoy a life of relative well being. Such therapy will not often effect a permanent cure. How much life will be prolonged is a point which will require further observation.

Case Reports

Sixteen cases of prostatic cancer have been treated by the author with castration and estrogen. The diagnosis was made by rectal palpation or biopsy or both. X-ray examination showed metastasis in five. The ages of the group ranged from 56 to 84 years. Eleven patients required at some time treatment for obstruction, usually by transurethral resection.

Castration was carried out immediately or after a few weeks of estrogen therapy except in four patients who refused operation because of senescence and a fixed obsession against surgery. Two of these

who refused operation have obtained striking relief from estrogens for periods of 16 and 22 months and are still living. However, they are beginning to show increase in the size of the prostate and their serum acid phosphatase levels are high. Of the twelve patients treated with castration, three were only slightly improved. Nine have made a splended initial response for a period of six months to fifteen months. Six died within a year after operation. Two cannot be traced. Regional deep x-ray therapy has been carried out in addition to endocrine therapy in several instances but its value has been very questionable.

The estrogen used after castration has been diethylstilbestrol in doses usually of 5 mg. daily. During the past two years I have used estinyl (ethinyl estradiol) in doses of 0.05 mg. to 0.5 mg. daily more and more.* It is the most powerful oral estrogen and provokes less nausea and perhaps somewhat less painful engorgement of the breasts.

CONCLUSION

Estrogen therapy has a definite but limited value in treating benign prostatic hypertrophy. There is suggestive evidence that its combination with testosterone at times may be of value. If favorable response is not definite and prompt and no contraindication exists, removal of the obstructing prostate by transurethral or incisional procedure is indicated.

Castration and estrogen therapy, however, give relief from pain and restore well being to the patient with prostatic cancer. They produce regression of the growth for a variable period of time. How long they actually prolong the life expectancy of the patient, in spite of an abundance of clinical evidence, remains a disputed point.

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DISCUSSION

DR. SAMUEL A. VEST, Charlottesville: Dr. Keyser has certainly covered a very wide field and a field which is most interesting to the male population over forty years of age, inasmuch as the incidence of either benign hypertrophy or cancer is extremely frequent. It increases each decade until at eighty years of age possibly eighty per cent of men show some degree of either one or the other. I should like to emphasize a few things about this condition and Dr. Keyser's paper.

In the first place, the therapy of any disease is much simplified if we know the etiology, but after much research and study of this condition the etiology is still obscure. For many years there was debate as to whether prostatic hypertrophy was due to arteriosclerosis of the arteries in the prostate or whether it is an actual tumor or simple hyperplasia. During the last decade we have gone through a hormone era; there has been discussion as to whether prostatism is due to too little body androgens or to an excess of female hormone or to female hormone deficiency or to an imbalance between the two. There is even a strong possibility of dysmetabolism by the liver, particularly of the female hormones. A lot of modern therapy has been based on animal experimentation, but I can safely tell you that the benign hyperplasia that Dr. Keyser has been talking about never occurs in animals. The hyperplasia that occurs in dogs is not the type of hypertrophy found in humans. The sum total is that a lot of therapeutic programs in humans have been carried out on theoretical experimental bases. I can quote you from the literature, from one clinic to another, in which clinical improvement has been reported as a result of such animal experimentation. In spite of all that, there is no scientific basis for rational therapy as far as animal experimentation is concerned. Therefore, such clinical studies as Dr. Keyser has given us will prove valuable in the future.

I think Dr. Keyser would be the very first one in this room to agree when I say that he belongs to merely one group on the use of estrogens in benign hyperplasia. I wish I could express a definite opinion myself, but we do not have the type of clinical material in our clinic that Dr. Keyser has been fortunate enough to have to treat large numbers of cases of benign hyperplasia with estro-

gens. I might also tell you that in other clinics opposite results from Dr. Keyser's have been reported. I am sure that Dr. Keyser would want you to know that. On the other hand, in other clinics there is considerable agreement with Dr. Keyser and an enthusiasm for estrogens.

Estrogens do not reverse the basic histology or produce any changes in the prostate which we know as hypertrophy. I think I can say from even our small series that there is sometimes a reduction in size, but that might be a result of reduction in the amount of secretion in the prostate and not reduction of the hyperplastic tissue itself.

The number of endocrine therapeutic possibilities are large. As to how much hormone to use, Dr. Keyser is on the band wagon for small dosage; others give larger amounts. Some conclude that estrogens by mouth are detoxified by the liver and urge injections as a method of administration.

There are many types of clinical studies in regard to hormone therapy being made. For example, at the University we wanted to know about the results of castration alone in cancer of the prostate. So we castrated about seventy-five cases. We put those cases aside in a closed group so far as statistics are concerned; and it will be possibly four or five years yet—because they live a long time—before all seventy-five are dead and we can then tell you that the average patient lived so many years. Another clinic may then have a report on other lines of therapy such as the results of small doses of estrogen, another one on large doses of estrogen, etc. I think out of all these studies perhaps in five to ten years we shall finally be able to have some idea of where we are actually going in this matter of hormone therapy in both benign hyperplasia and in cancer, and only then will we be able to correctly evaluate the improvements.

We have not yet found the complete answer, but such data as Dr. Keyser presented should be recorded. It will prove valuable to us not only now but in the future, and I wish to thank him personally for it.

DR. KEYSER, closing the discussion: I wish to thank Dr. Vest for his excellent discussion and the well considered opinions he has expressed on this controversial subject. Several points should be emphasized.

As stated in the body of the essay, the experimental work done on animals is very inconsistent and somewhat confusing. In any research problem with a background of animal experimentation the evaluation of the therapeutic merit suggested thereby must await clinical trial.

With regard to benign prostatic hypertrophy, when I started treatment with hormones I had an initial series of rather brilliant results and—perhaps unfortunately—became enthusiastic over this form of therapy. I am still enthusiastic over its possibilities in selected cases. Certain patients with large glands and definite obstruction do obtain what seems to be a result comparable with that afforded by prostatectomy. Unfortunately this is the exception rather than the rule.

Also, it should be brought out that early cases of carcinoma amenable to radical surgery may be missed in using hormone therapy. About 20 per cent or more of all prostate glands removed show histologic malignancy.

There is a psychological factor in dealing with older men which is of tremendous import. They are hoping to find the fountain of youth. While this is not to be entirely discouraged, we must keep a scientific attitude and not become too enthusiastic.

I am in agreement with Dr. Vest that any therapy along this line must be well thought out and well planned. Finally, and I am certain Dr. Vest would agree, any treatment of prostatism is a matter to be carried out or at least carefully supervised by a well trained urologic specialist. This is especially true of hormone therapy. It should be emphasized that surgery is still by all odds the treatment of choice in prostatism. The general use of hormone treatment by those not trained in urology would bring such therapy into disrepute and cast doubt upon whatever merit it may have in selected cases.

Floral Eponym
Galtonia

SIR FRANCIS GALTON, 1822-1911

Sir Francis Galton, a cousin of Charles Darwin, was born at Birmingham and received his education at Birmingham Hospital, at Kings College, London, and at Trinity College, Cambridge. During 1845-46 he traveled in the Sudan and in 1850 he explored South-west Africa, and on his return he published *An Explorer in Tropical South Africa*. Later he became interested in meteorology and was the first to chart the weather on an extensive scale. In 1859 he began to study anthropology, heredity and the application of statistics to human attributes. He is considered the founder of eugenics.

Galtonia is a small genus of South African bulbous herbs of the lily family. The best known is G. candicaus or summer hyacinth.

XUM

MENINGITIS IN INFANCY*

WM. B. MCILWAINE III, M.D.,
and
HERMAN FARBER, M.D.,
Petersburg, Virginia.

The object of this presentation is not to discuss the subject, meningitis, in all of its many ramifications, nor to report any large series of cases, nor to bring forward some new treatment. It is simply to recall, to us all, the fact that meningitis in infants does not present the clear cut, text-book picture we expect, and so often find in older children, and the importance of considering the meninges as a focus of infection in all cases of unexplained illness in infants even though the symptoms and signs are meager or totally non-existent.

In my own experience it is rather startling, after practicing pediatrics for over twenty-five years, to have some new knowledge which makes me wonder how many times I may have overlooked until too late, a condition which now I feel sure I shall remember for the next twenty-five years (I hope).

Dr. Farber while studying under Dr. H. D. Long in Chattanooga recalls the admonition given by Dr. Long that in any case where an infant runs an unexplained fever for 48 hours do a lumbar puncture.

In discussing the diagnosis of meningitis in infancy Drs. Hogg and Bradley,[1] while reporting a case of pneumococcus meningitis in the new born with recovery, review the literature and quote Dr. Cooperstock as saying that "unexplained fever may be the only sign, and with convulsions should make one suspicious of meningitis." Cooperstock[2] himself, writing in the *Journal of the Michigan Medical Society* in 1938, says, "out of 12 cases of meningitis in infants admitted to the clinic only one was diagnosed before admission." I also quote Cooperstock as saying, "Instances are met with in which unexplained fever exists as the solitary evidence of meningitis in which examination of the spinal fluid holds the final answer."

Dr. J. M. Ravid,[3] writing in the *American Journal of Diseases of Children,* quotes McLeen and Caffrey as saying, "Meningitis was not recognized in 136 infants prior to entrance to the hospital and in some cases the diagnosis was not reached even after many days in the hospital."

*Read before the annual meeting of the Medical Society of Virginia in Richmond, October 18-20, 1948.

Ravid himself states that "tension and bulging of the fontanel is always present and is one of the most reliable signs of meningitis in infancy." He also considers that unexplained fever with diarrhea which does not respond readily to treatment requires a spinal puncture.

Herman,[4] writing in 1915, in reporting a case of meningitis in the new born, writes "No meningeal symptoms were present at any time, yet the autopsy showed a purulent meningitis and the culture a pure culture of pneumococci.

Koplik,[5] writing in 1916, gives the symptoms and signs of meningitis in the young infant as fever, twitching, and a peculiar groaning respiration, and stresses the fact that there may be no irritative symptoms nor rigidity but generally the infant is quite limp with cyanosis and marked apathy.

In the 14 cases we are reporting we have tried to outline the symptoms and signs and diagnoses as follows:

CHART NO. 1

As will be noted, all of our cases had fever. While only the admission temperature is given, practically all ran a rather moderate, irregular febrile course except for a rapid rise to 105-107 as a terminal event in those who died. Only four of our cases had convulsions, a condition so often and freely associated with meningitis. In only six cases was vomiting a noticeable symptom, but, along with six cases in which stupor was present in varying degrees, was the most constant finding other than fever. Only three cases of the 14 presented signs of meningeal irritation and in only three cases was a rash, generally petechial in character, noted. In three cases pallor was a striking symptom, although most of these babies were pale and showed a moderate anemia by the routine blood counts.

An impressive fact was that in not a single case was tension or bulging of the fontanel noticed, which leads us to believe that this sign must be more frequently seen in the new born than in infants after the first month of life. Eight cases had complicating diseases, three of which was pneu-

CHART No. 1

NAME	AGE (mo.)	RACE	SEX	FEVER	CONVULSIONS	VOMITING	STUPOR	SIGNS OF MENINGEAL IRR.	BULGING FONT.	RASH	PALLOR	COMPLICATIONS	TYPE	RESULTS
1. B.H.	1	W	F	101							x		M	R
2. D.W.	2	W	F	102								Pneumonia	P	R
3. J.T.	2	N	M	102								Diarrhea	P	R
*4. L.W.	2	W	F	104.8								Pneumonia	?	D
5. N.H.	2	N	M	99.8	x								S	D
6. H.W.	4	W	M	101			x				x		M	R
7. C.M.	6	N	M	99.2			xxx	x					P	D
8. R.H.	6	W	M	101		x	xxx			x			M	D
†9. R.B.	8	W	M	104.4	x	x	xx					Otitis Media	P	R
10. N.G.	9	W	F	103.4	x	x	xxxx				x	Pneumonia	P	R
11. L.L.	9	W	M	100.4								Lt. Side Paralysis	?	R
12. G.E.	11	W	F	102.6		x				x			P	D
13. M.M.	12	N	F	98		x		x				Lues	M	R
14. A.M.	12	W	M	103.2	x	x	xx	x		x			?	D
NAME	AGE	RACE	SEX	FEVER	CONVULSIONS	VOMITING	STUPOR	SIGNS OF MENINGEAL IRR.	BULGING FONT.	RASH	PALLOR	COMPLICATIONS	TYPE	RESULTS
Totals		10 W 4 N	8 M		4	6	6	3	0	3	3	7	4 M 6 P 3 ? 1 S	6 D 8 R

*Dr. Læta J. White.
†Dr. William Grossmann

monia. Two of these may have been the cause of the meningitis and one may have been a complication of the meningitis.

In only one of our cases was diarrhea or gastro-intestinal disorder noted, which does not correspond with the view of Ravid.

Paralysis occurred in only one case.

Lues, noted in one case, seemed to be of no importance in the course of the meningitis.

One case very definitely was a direct extension

from a purulent otitis media and mastoiditis.

The spinal fluid was under increased pressure in all cases, varying from slightly cloudy in two cases to purulent in one case. The cell count was above normal in all cases with a wide range of variation from 10 to 88,200.

The organism causing the disease was the meningococcus in four cases, one of which died. The pneumococcus was the causative organism in six cases, of which two died. The influenza bacillus was the causative organism in only one case which recovered. The streptococcus was the causative organism in one case which died, and in two cases the causative organism was not satisfactorily established, one of which recovered and one of which died. In all there were eight recoveries and six deaths.

CASE REPORTS

Case 1.—B. H. was a one month old white female. She lived in a rural community. The environmental and sanitary conditions of her home were good. The parents were intelligent and at all times cooperated with the physician.

History of the Present Illness.—On Oct. 19, 1946, the infant was seen for the first time at home. The mother said that the baby had been having a slight fever for about 24 hours. Otherwise the mother had noticed nothing wrong with her. Examination at this time did not show any cause for the fever. There was no evidence of any upper or lower tract respiratory infection. The temperature was 101. The baby did not appear to be very ill and was actually playful, but was given small doses of aspirin. The next day, Oct. 20, the infant still showed the same low grade fever (101). Physical examination still did not show any cause for the fever. At this time particular attention was paid to the examination of the nervous system. No pathologic or abnormal manifestations could be made out from the neurological examination. The infant was started on sulfadiazine by mouth. At this time it was noted that the baby was probably a bit more drowsy than the temperature would indicate. On Oct. 21st, the baby was seen again at home. A profound change had taken place. The infant was very drowsy. It could be aroused but would immediately drift back into a deep sleep. The temperature was still 101. There were no other signs suggestive of meningitis. The tension of the fontanel was normal. The infant appeared much paler than it had on the previous day, and was admitted to hospital.

Physical Examination.—This revealed a baby in a deep sleep. Examination by systems was negative. The neurological examination was normal. The baby was pale. The tension of the fontanel was normal.

Laboratory Studies.—Spinal fluid showed 6,500 w.b.c. predominantly polys. The sugar was decreased. The smear showed many gram negative intracellular diplococci.

Course in the Hospital.—The temperature never went over 102. The baby developed classical signs of meningitis as manifested by stiff neck and a Kernig within a few hours after the first lumbar puncture. She was started on penicillin intrathecally and intramuscularly. She was also given oral sulfadiazine. The neurological signs disappeared slowly on this infant. She retained a stiff neck for about two weeks. At times she would have projectile vomiting. The infant remained drowsy for about 10 days. She always took her nourishment well and feeding her was never a problem. She was discharged in good condition on Nov. 11, 1946. She has been seen several times since then and has shown no complications or sequlae as a result of the meningitis.

Case 2.—H. W. was a 4 months old white male. Admitted Feb. 9, 1948, and discharged on Feb. 20, 1948. He lived in a rural community and the sanitary and evironmental facilities of his home were very bad.

History of the Present Illness.—The baby was put to bed feeling fine on the night of Feb. 8 (the night before admission). The baby did not awaken at the usual time the next morning. When the mother went into the room to see why the baby had not awakened, she noticed that it felt hot and was breathing rapidly. She also noticed that the baby was pale. She became frightened and brought the infant to the hospital.

Physical Examination.—This revealed a very pale baby. The respirations were rapid. The baby was drowsy but could be aroused without much difficulty. Examination of the body by systems was normal except for the extreme pallor and the difficult respirations. There were no *positive neurological findings.* Tension of the fontanel was normal.

Laboratory Studies.—R.B.C. 2,810,000; Hb.

60%; W.B.C. 10,500. Differential was normal. X-ray of the chest was normal. Lumbar puncture Feb. 9, 1948, showed a cloudy spinal fluid. The cell count was 50 and showed 100% polys. The smear showed many gram negative intra and extracellular diplococci.

Course in the Hospital.—The baby was given sulfadiazine by mouth and penicillin intrathecally. The baby's temperature never went over 101 degrees during the entire hospital stay. He never developed any classical signs of meningitis such as stiff neck or positive Kernig. The baby improved from the beginning of the therapy. The infant was given 2 blood transfusions to combat the anemia. No difficulty was experienced with vomiting. The baby was discharged on Feb. 20th with no apparent sequelae or complication of the meningitis.

Case 3.—N. G. was a 9 months old white female. Admitted to the hospital Dec. 15, 1946. She lived in a rural community. The environmental and sanitary conditions were not good.

Past History.—This baby had been ill with a fever for about one week. The history that could be obtained was unreliable. The mother had been treating the fever with aspirin. She thought the infant had only a cold. On Dec. 15, 1946, the baby had a severe generalized convulsion.

Physical Examination. — Examination at this time revealed a well developed white female baby. The color was pale. It was evident that the infant was quite ill. The respirations were rapid and labored. At this time the patient was having a generalized hard convulsion. The temperature at this time was 103. There were signs of a rather extensive pneumonia on the right side. Examination by systems of the other parts of the body did not reveal any pathologic or abnormal findings. The neurological examination did not show any abnormalities.

Laboratory Studies.—W.B.C. on admission showed 22,000, most of which were polys.

Course in the Hospital.—The baby was started on penicillin as soon as it was admitted to the hospital. The convulsion was treated routinely with cool enemas, cool sponges, sedation and aspirin. It was thought that the temperature was higher just before admission to the hospital. The mother said that it felt much "hotter" at home and she had given the baby a half an aspirin just before bringing her to the hospital. The convulsion lasted only a minute or two after being admitted. The baby seemed to be much sicker than the temperature would indicate. The patient was taking nourishment well and seemed to be gradually getting better. The signs of pneumonia were gradually clearing up. On Dec. 22, the nurses noted that the baby had difficulty in keeping its food down. It would vomit a few minutes after eating. The vomiting was non-projectile in character. The temperature at this time was not over 100. However, the baby continued to look well and did not show any evidence of dehydration. On Dec. 24 the temperature was 102. The infant was still vomiting of a non-projectile type. *A lumbar puncture* was done. It showed *675 cells* most of which were polys. The globulin was 4 plus. There were many *gram positive diplococci* present both intra and extracellular. The culture of the spinal fluid was *positive for pneumococci.* The patient was given intrathecal penicillin, sulfadiazine by mouth, and intramuscular penicillin. The intramuscular penicillin was never discontinued from the onset of the diagnosis of the pneumonia. The baby was given 3 blood transfusions to combat the anemia that developed after the diagnosis of meningitis. The baby never developed any of the classical signs of meningitis as manifested by stiff neck, Kernig's, etc., and was discharged in good condition on Jan. 9, 1947.

SUMMARY

1.—Fourteen cases of meningitis in infants from 1 to 12 months of age inclusive have been reported.

2.—The lack of uniform symptoms and signs and at times the absence of any symptoms or signs of meningitis other than fever has been noted.

3.—The advisability and importance of early spinal puncture for diagnosis has been stressed.

4.—Three cases illustrating the subject matter of the report have been discussed in detail.

5.—We feel that today, as has been and always will be, the question of diagnosis is of paramount value both to the patient and the physician.

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XUM

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DISCUSSION

DR. EDWIN L. KENDIG, JR., Richmond: I have enjoyed very much the discussion of the problem of meningitis in infancy by Dr. McIlwaine and Dr. Farber, and I appreciate the privilege of discussing it.

Actually, I have little to add, but there are two points that I should like to emphasize. Certainly this paper has shown again that the usual signs and symptoms of meningitis in older children and adults are not necessarily present in the disease as it appears in infancy.

Since early diagnosis is of paramount importance in the establishment of successful treatment of the disease, it would seem logical to say that any febrile disturbance in infancy, the cause of which is not immediately apparent, deserves a lumbar puncture. Further, spinal puncture should be done on any infant who has a convulsion.

DR. ———: I enjoyed very much Dr. McIlwaine's description of meningitis in infancy. I think, as has been manifested both by Dr. McIlwaine and by the discusser, you cannot make a diagnosis of meningitis in an infant that young by the physical signs alone. We have had occasion to treat about five hundred cases of influenzal meningitis and about forty cases in infants in our hospital in the last five years. I think one thing to be emphasized is not only to make a diagnosis of meningitis but also of what type of meningitis it is if you are going to get a rational therapy.

Nothing has been brought out about the treatment of meningitis, except in a vague way. It is our policy in treating influenzal meningitis to give them the whole works. Dr. ———, in New York, thinks streptomycin alone is enough. In our cases of meningitis, of which I have treated forty, with about four deaths, in the last five or six years—we treated practically all with a combination of Type B influenza, saline, sulfadiazine intravenously, and also streptomycin intramuscularly. In the last few cases we have not given any streptomycin at all and think that penicillin intrathecally is uncalled for and unnecessary except in streptococcic meningitis. In influenzal meningitis I give the streptomycin, penicillin, and sulfadiazine intravenously. We have had a few over forty cases, with four deaths.

DR. MCILWAINE, closing the discussion: I want to thank Dr. Kendig and Dr. ——— for their discussion. We did not discuss any therapeutic measures, and I am very grateful to the doctor for giving us the last word on it.

Of course, I think the less you have to go into the spinal canal the better. If we can treat meningitis by oral medication and the intramuscular and intravenous routes it will be more satisfactory.

Heart Infection Takes Heavy Toll in Disability.

Despite the success doctors have achieved in curing infection of the lining of the heart by administering penicillin, patients who recover from the disease may be disabled.

One out of three patients in a group of 18 reported in the September 10 *Journal of the American Medical Association,* were left with a progressive heart condition, although penicillin cleared up the active infection. Subacute bacterial endocarditis, inflammation of the membrane which lines the heart, has been until recently an almost uniformly fatal disease. In a number of cases it follows rheumatic fever.

With the advent of penicillin therapy, however, doctors have been able to cure many patients of the active heart infection. But since the membrane which lines the heart muscle covers the valves of the heart as well as its inner walls, endocarditis may leave scars which cause narrowing of one or more valves or interfere with their proper closing.

All of the group of patients reported by Drs. Sherman R. Kaplan, Ray H. Rosenman, Louis N. Katz, and William A. Brams, of Michael Reese Hospital, Chicago, were followed from 25 to 61 months after their heart infection was cured by penicillin therapy. Six of the patients had progressive heart disability since the onset of subacute bacterial endocarditis. In three of these the disability led to death from heart failure. Twelve showed no progression of their heart condition, the doctors say.

XUM

PRE-OPERATIVE STUDY OF THE MALFORMED NOSE

WM. LAWRENCE GATEWOOD, M.D.,
Richmond, Virginia.

To properly correct the deformed nose, adequate appraisal of all the parts involved in the production of the malformation is required. This can only be accomplished when a painstaking examination of the external and internal nose is made and an analysis of the findings, resulting from this examination, has been carefully noted. A thorough anatomical study of the nose is of paramount importance to establish every anatomical constituent in the etiology of the malformation and before proceeding with a description of the method of examination, it would be well to review briefly the principal anatomical structures.

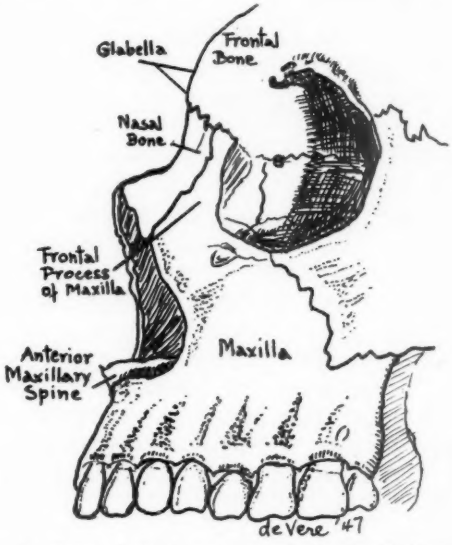

Fig. 1. Bones of the face—in the nasal and infratemporal regions.

CLINICAL ANATOMY

The nose is composed of bone and cartilage covered by skin externally and mucous membrane internally. The septum bisects the nose longitudinally, separating the two nostrils. Structurally from above downward, the nose is composed of a bony vault, a superior cartilaginous vault and an inferior cartilaginous vault (Fig. III). Each of these structures form a part in maintaining the bridge line and the contour of the lateral walls and each is dependent on the septum for support (Fig. II).

Bony Vault.—This is composed of the two nasal bones; they rest on the frontal processes of the maxilla and join each other in the midline, producing

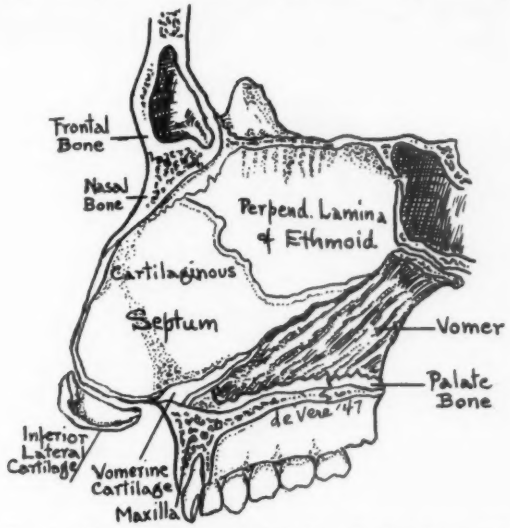

Fig. 2. Lateral view of nasal septum—showing bones and cartilages that form its skeleton.

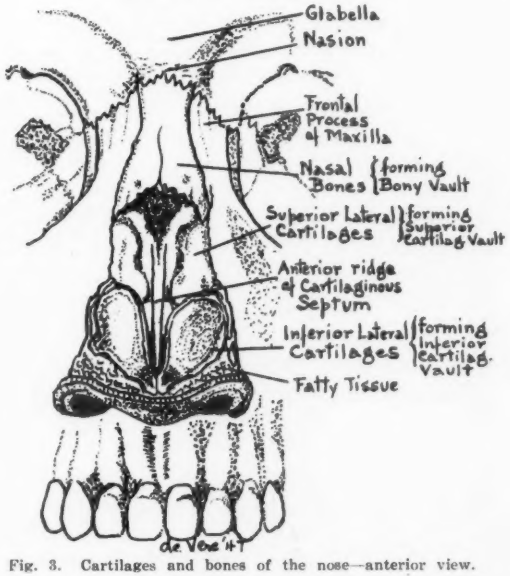

Fig. 3. Cartilages and bones of the nose—anterior view.

what is known as the bony dorsum or bony bridge (Fig. III). The nasal bones also obtain attachment to both the bony and cartilaginous parts of the septum. Their upper borders are attached to the frontal bone with which they form the root of the nose. On the dorsal line, this point is known as the glabella. Here, the two nasal bones are slightly separated to allow the interposition of the strong internal angular process of the frontal bone. The lower free borders of the nasal bones receive the superior lateral cartilages which are attached to them by fibrous bands (Fig. III and IV).

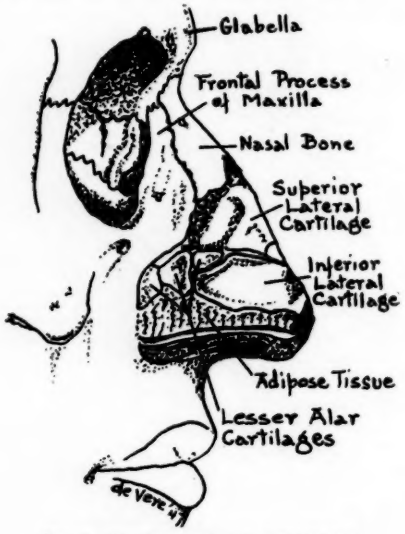

Fig. 4. Cartilages of nose—lateral view.

Superior Cartilaginous Vault.—This consists of two triangular pieces of cartilage placed on either side of the cartilaginous septum. The long side of each is fixed to the septum, the upper edges to the nasal bones, and the lower edges to the lateral crura of the inferior cartilaginous vault. These cartilages are known as the superior lateral cartilages (Fig. III, IV and V). They are responsible for the contour of the middle third of the lateral nasal walls and dislocation of their septal attachments produces asymmetrical convexity or concavity in accordance with the anatomical disorganization which follows.

Inferior Cartilaginous Vault.—This is composed of two V-shaped cartilages, each of which frames its respective nostril. The angles of the two cartilages form the nasal tip. The medial crus of each, attached firmly to its fellow by fibrous tissue, makes up the so-called membranous portion of the septum. This is contiguous with the lower border of the cartilaginous septum and provides a support for the skin of the columella. The medial crura, about ½ cm. wide, end posteriorly in

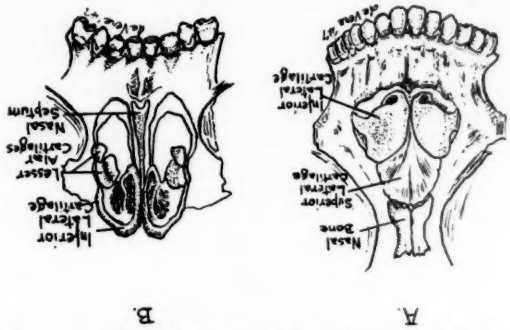

Fig. 5. Cartilages of the nose—"A" anterior view, "B" inferior view.

slight lateral deviations which run beneath the floor of the nostril to gain attachment to the nasal spine. The lateral crura curve outward from the nasal tip to give support to the nostrils before ending in the region of the alae (Fig. II, III, IV and V).

Septum.—This consists of bone and cartilage. The bony septum is composed of the vomer and the perpendicular plate of the ethmoid. The ethmoid rests on the vomer. The cartilaginous septum is quadrangular in shape and situated in the lower half of the nose. It rests on the palatal processes of the maxilla and on the vomer which is grooved to receive it (Fig. II).

EXAMINATION

External Nose.—The nose should be examined full face and in profile and should be compared with photographs of the patient taken before the

A. Congenital malformation of nose. After surgical reconstruction.

B. Posttraumatic malformation of nose. Post Operative result. Posttraumatic flattening of lower aspect of nose. After surgical repair.

onset of the deformity, if these are obtainable. When examining the full face or the profile, each of its three component parts, the bony vault, the superior cartilaginous vault, and the inferior cartilaginous vault must be studied to obtain an accurate assessment of the anatomical displacements. When viewed full face, the nose may be obviously deflected from the midline. The point of maximum deviation must be determined by laying a ruler over the root of the nose and center of the upper lip. This is readily seen and the position of the nasal tip revealed relative to the midline. From the same viewpoint, abnormal breadth of the nose, either in its bony or cartilaginous parts, is at once apparent. The region of the superior lateral cartilages is examined for abnormal sagging or undue bulging and the nostrils are examined for asymmetry and inequality of size.

Unilateral prominence of the nasal tip is noted. Flattening of the alar region, indicative of separation of the attachment of the inferior lateral cartilage from the maxilla, is looked for. Palpation of the lateral nasal walls confirms any suspected irregularity of the bony contour, demonstrating bony elevations, overlapped comminuted fragments and sometimes an infracted shelf of the nasal bone still attached to the maxilla. When seen in profile, an irregularity of the bridge line is at once in evidence. Usually, such an irregularity consists of a depression and careful note is made of location of the maximum collapse. Occasionally, an irregular prominence is seen on the dorsal line bordering the depression. This is due to the elevation of the opposite end of a sliver of a slithering bone tilted as it became displaced backward when the bridge collapsed. Examination in profile will also reveal any abnormal retraction of the lateral margin which is confirmed by comparing the two sides. With the patient's head well extended backward, the columella is visualized and any abnormality, such as widening and deviation, is recorded.

Internal Examination.—An estimation of the degree of obstruction of the airway is first made by instructing the patient to inspire and expire through

C. Gross posttraumatic distortion of nose. After correction. Posttraumatic broadening of entire nose. After surgical correction.

XUM

each nostril. The mucosa is then inspected with the aid of a speculum and good light. When septal deflections are significant, special note is made of their relationship to the bridge line and to the lower border of the septum. Careful examination is made to decide whether the deviation is confined to either the bony or cartilaginous parts or whether both of these structures are involved. Bridging of the lateral and medial mucosal linings is looked for and is often found to be extensive. With a fine probe, the extent of such adhesions is investigated and the amount of underlying bony block determined. Spicules of bone or cartilage, driven through the mucosa at the time of injury, may still be found to be incompletely covered. Finally, the region of the middle turbinal bones is examined on both sides of the nose for evidence of purulent discharge from the sinuses. If sinus disease is suspected, other diagnostic measures should be resorted to, including x-ray films of the sinuses.

Clinical Photography

Before operative treatment is begun, clinical photographs of the nose should always be taken. Full face, right and left profile views are essential. For the purpose of case records, a series of clinical photographs is extremely informative and, together with detail notes, form a backlog of knowledge which is so much needed at the operating table. It is important to have a standardized technique so that each picture can be compared with those previously taken and the best results are obtained if the same technician is able to take all of the pictures in the series.

The examination outlined should always precede the necessary surgical technique which must be meticulously planned in advance of the actual operative procedure. The purpose of surgical treatment in deformities of the nose is to restore the nose to its correct shape, thereby improving both its appearance and function. This requires free mobilization of all misplaced parts and the necessary reconstruction to properly shape the bony and cartilaginous structures. The operation should effect the shortening of the elongated nose, straightening of the crooked nose, narrowing of the broadened nose, flattening of the humped nose, elevation of the saddle nose and the adjustment of the set and lie of the columella and the superior and inferior lateral cartilages. Mobilization of the nasal bones and severance of their attachment to each other and to the septum is often required.

The method of examination described should be applicable in the study of the various types of malformations of the nose, congenital or acquired, with the exception of those cases of total or partial loss. Deformities, due to trauma and disease, occur less often than do those of congenital origin and they not infrequently present interesting and difficult problems in the matter of their correction.

In undertaking the surgical repair, it must be remembered that the nose, being the most conspicuous facial feature, challenges attention if it materially differs from the means of the normal nose. As disguise of the deformity is impossible, surgery remains the only recourse and such reconstruction must conform to the other features of the face or its purpose will be defeated.

Comment

To obtain a satisfactory esthetic and functional result, a thorough preoperative evaluation of the deformity is essential and the plan for the necessary operative steps should be based upon the findings, resulting from this pre-operative study. It is obvious that good surgical craftsmanship, executed through intranasal incisions, together with artistic, creative power on the part of the surgeon in shaping and reassembling the structures involved, should materially enhance the outcome.

The accompanying photographs show post-traumatic and congenital deformities of the nose before and after surgical reconstruction.

Note the more youthful appearance of the patient subsequent to surgical repair.

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ETIOLOGICAL FACTORS IN PANCREATITIS*

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An understanding of the fundamental steps taking place in the development of any disease is necessary in order to prescribe and carry out the proper treatment. Our system of medicine requires a diagnosis, and we feel that only with a diagnosis can we offer the large number of patients relief. Treatment is directed toward alleviation of causative factors.

Pancreatitis falls into that group of morbid processes which we do not understand thoroughly. A review of the etiological factors is proposed, and suggestions toward a physiological plan of therapy will be presented.

Diseases of the pancreas were almost unknown until Reginald Fitz in 1889 gave us a classification of pancreatitis. The anatomy of the pancreas is important because of its relations and its retroperitoneal position, lying at the level of the first lumbar vertebra. The head is surrounded by the duodenum, the lesser sac and celiac axis above, the superior mesenteric artery below. The tail is in relation to the splenic artery and the hilum of the spleen. The numerous arteries are derived from the hepatic, splenic, and superior mesenteric arteries, giving it a very rich blood supply. The veins are part of the portal system passing into the liver. The main duct (Wirsung) drains the entire pancreas, and empties into the duodenum in relation to the common bile duct. The accessory duct of the head (Santorini) usually has an independent opening.

The nerve supply from the splanchnic plexus, consists of secretory fibers from the vagus nerve, and vaso-motor fibers from the sympathetic trunk. Stimulation of the vagus nerve yields a thick fluid rich in enzymes. This action is blocked with atropine. Further secretions, chiefly water and inorganic substances, follow injection of the first hormone, secretin. Further secretion also is evoked by the presence of acid gastric chyme in the duodenum. Stimulation of the sympathetic fibers may result in an increased secretion, but this is thought to be a vascular effect.

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The chief enzymes are trypsin, lipase (steapsin) and the amylases. Trypsin is activated by hydrochloric acid, bile, or calcium salts and splits protein into peptones, and peptones into amino-acids. It works best in an alkaline media, while the proteolytic gastic pepsin works best in acid media. The lipase splits the fat into fatty acids and glycerin, aided in the emulsification by bile salts. Many amylase varieties are present, splitting starch and all of its sugars. Measuring increased amylase activity in the blood is the most common means of determining the presence of pancreatitis.

The morbid anatomy characteristic of pancreatitis is the development of a diffuse glassy edema of a swollen indurated gland or a hemorrhagic necrosis, followed by an abscess formation in more severe grades. Fat necrosis on the surrounding surfaces and hemorrhagic peritoneal exudates are common findings. The cause of this change remains incompletely answered.

It is definitely accepted that a "common channel"[1] is the most common anatomical finding necessary to bring about pancreatitis.[2,3,4,5,6,7] While bile can normally be present in the pancreatic duct without incident, some change in its character or method of entry activates trypsinogen. Ductal obstruction[8,9] from any cause is a precipitating factor. Doubilet has prevented recurrent pancreatitis by section of the sphincter of Oddi.[7]

Experimental evidence showing the severe results following induction of pancreatitis in an actively secreting gland have recently been published,[9,10] bearing out clinical opinions of observers the past sixty years. Rich and Duff[8] felt that the degree of trypsin activity determined whether the gland would show edema or hemorrhagic necrosis. Popper and his associates[5] were unable to produce hemorrhagic necrosis in acute pancreatic edema until they temporarily obstructed the pancreatic artery for fifteen minutes. This anoxic factor is very significant, though the degree is not clear to most interested clinicians. It is suggested that relief may come from release of vaso constrictive impulses by procaine splanchnic anesthesia.

XUM

Infection of the pancreas was the earliest described cause of pancreatitis, the mode of approach being duodenal, ductal, lymphatic, or hematogenous. This is not held to be a factor now, but, when present, is secondary to the necrosis. This is all that can be seen from a pathologists' point of view.

Alcoholism, as a factor, probably causes a duodenitis with spasm of the sphincter causing a reflux.

Anaphylaxis will be mentioned only because it has recently been called to attention in the literature.[11]

Treatment based on consideration of the etiological factors consists of conservative methods immediately, and operative methods later. First, relief of the intense pain with morphine or demerol is always covered by simultaneous injection of atropine. I prefer an immediate splanchnic anesthesia using Labat's modification of Kappis' approach with 1% procaine.[12] It has been noted that the severe pain of pancreatitis seldom recurs following splanchnic anesthesia, while the severe colic of gall stones, renal stone, and intestinal obstruction nearly always recurs.[13] Relief of severe pain seen in pancreatic lithiasis and chronic pancreatitis not only follows the radical procedure of pancreatectomy, but also follows splanchnicectomy.[14,15,16] Clinical trials with procaine splanchnic anesthesia not only demonstrates the relief of pain but a definite therapeutic effect too.[17]

Second, immediate gastric suction is started, and intestinal motility and secretion is kept at a minimum for days. Guides to removal of the tube are normal passage of gas by rectum, a normal pyloric balance, and the disappearance of the upper abdominal tenderness.

Third, intravenous fluids, as needed, and for replacement, include blood, amino-acids, glucose in water, and isotonic saline.

Fourth, close observation for the development of complications must be carried out. Diabetes, localized suppuration, pancreatic cysts, and hypocalcemic tetany have been described, and are more common as the severity of the lesion increases. Insulin, proper surgery, and calcium serve to ameliorate these developments.

Fifth, determination of the precipitating factors, if possible, must be carried out. As a general rule, cholecystitis with stones will be found, and cholecystectomy, choledochotomy, and endocholedocal dilatation of the sphincter of Oddi and ampulla can be carried out. Bakes dilators up to 8 or 9 mm. generally suffice. In treating recurrent acute pancreatitis, Doubilet and Mulholland[7] have found it necessary to cut the sphincter of Oddi in 21 instances with relief in 20, in order to have adequate and free flow of the biliary pancreatic secretions. Allen and Wallace, showing the simplicity and effectiveness of Bakes dilators,[18] described the finding of the ampulla of a patient of Dr. Bakes 76 days post-operatively, when the number 13 sound was easily passed. No cicatricial tissue was found; the patient died following severe gastric bleeding, unrelated to the biliary disease.

SUMMARY

Pancreatic duct obstruction from ampullary stone, stricture, or spasm, or from inspissated mucus or metaplasia of the duct epithelium appears to be a prime precipitating factor in acute pancreatitis.

Trypsinogen activation, which follows, accounts for a large amount of the pathology.

Local ischemia from vaso-spasm with the resulting anoxia may be the factor which lowers the normal resistance of the pancreas to enzyme damage.

An actively secreting gland increases the severity of the lesions experimentally and clinically.

CONCLUSIONS

The role of vaso-spasm and anoxia in pancreatitis must be more clearly understood.

Recognition of the concept of obstruction as the prime factor in precipitating acute pancreatitis, leads to a surgical attack on the ampulla of Vater to prevent recurrences following indicated cholecystectomy.

Splanchnic anesthesia appears to have definite therapeutic value in early management of acute pancreatitis, in addition to relieving pain.

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183 North Upper Street.

Keep Moth Balls Away From Children.

Sucking moth balls can cause severe poisoning in children, warn two Detroit doctors. Four cases in which two-year-old youngsters were made acutely ill by eating moth balls of naphthalene are reported in the September 17th *Journal of the American Medical Association* by Drs. Wolf W. Zuelzer and Leonard Apt of Wayne University College of Medicine.

The substance caused an anemic condition. All four children recovered completely after receiving blood transfusions and other treatment.

"In each of the four patients, a definite history of naphthalene ingestion was obtained, although the exact amounts could not be established. Each child previously had been in perfect health and had not been exposed to other chemicals with hemolytic action [destroying the red blood cells]. Within a period varying from three to seven days, severe anemia developed in each case. The patients eventually made a complete recovery with return of the blood values to normal levels. All of our patients were approximately two years old, an age group particularly prone to the ingestion of household poisons. The symptoms begin with listlessness and loss of appetite followed by vomiting and other gastrointestinal disturbances. Eventually pallor is noted. Since moth balls are a common and easily accessible household article, generally believed to be harmless, and since the American literature seems to contain no previous reports of this kind, the cases are reported in detail."

The doctors do not suggest that any first aid procedures to combat moth ball poisoning be carried out at home before a doctor is called.

XUM

OPTOCHIASMATIC ARACHNOIDITIS REPORT OF A CASE*

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Since the first description of a localized process of arachnoiditis in 1898 by Schlesinger, and the first operated case in 1903 by Spiller, Musser and Martain, the entity of optochiasmatic arachnoiditis has become a recognized syndrome in diseases of the optic nerve and chiasma. The first surgical case was reported in 1929 by Balado. The thorough review of the literature by Ballock in 1939 numbered 129 reported cases. Up to the present date the writer has found a total of 164 cases of optochiasmatic arachnoiditis which have been diagnosed with or without surgical confirmation.

No single entity can be attributed to the formation of optochiasmatic arachnoiditis. In many cases the etiology is never determined. Infection of the meninges and brain by syphilis, trauma, sinusitis, petrositis, mastoiditis, and chronic rhinopharyngitis have been listed as causative factors.

Optic atrophy is the most frequent associated condition in these cases; however, a normal fundus may be seen.

The following most characteristic features suggesting the diagnosis of optochiasmatic arachnoiditis are:

1. Rapid loss of vision, at first with normal fundi and later with simple optic atrophy.

2. Visual field changes, frequently central scotomas, early concentric contraction of the fields; bitemporal hemianopsis is rare or late.

3. Mild evidence of infection and an episodal rather than a steady progressive loss of vision.

4. Roentgenological Evidence: The sella is normal, and there are no osseous defects of this region. Air encephalography usually demonstrates a deformity of the chiasmatic cistern. The air shadows are not clear; occasionally the cisterna interpeduncularis and pontis are absent, deformed or encroached upon and structures usually seen within the cistern cannot be identified, as demonstrated by Pendergrass.

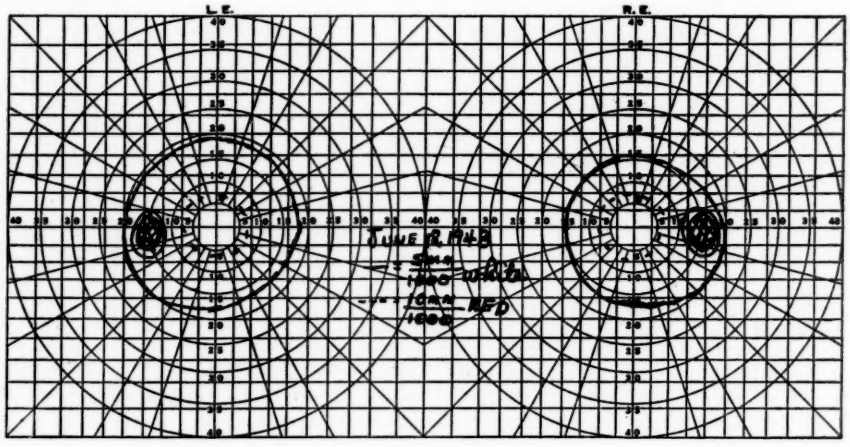

Fig. 1. (Raiford) Central fields showing generalized contraction. Vision OD 2/60. OS 2/60

5. Absence of signs of pituitary insufficiency.

6. Transfrontal headaches, and those of the retro-orbital type are present in about one-third of the cases. This usually is transitory and occurs in the earlier part of the disease.

The pathological findings are variable, from thin bands to that of arachnoiditis covering the entire

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area in a thick layer. Circumscribed pockets of serous meningitis with cyst formation are seen, which may simulate a tumor mass. The optic nerves may be flattened or reduced in calibre; the color reveals atrophic changes.

The greatest frequency is from twenty to forty years of age, with a total of seven cases reported less than ten years old.

Medical treatment has been rather disappointing in these cases. The surgical approach has rendered the best therapy and should be done as early as possible in order to preserve the visual nerve elements.

CASE REPORT

The patient was an eight-year-old white male, admitted to the Graduate Hospital of the University of Pennsylvania on June 15, 1948.

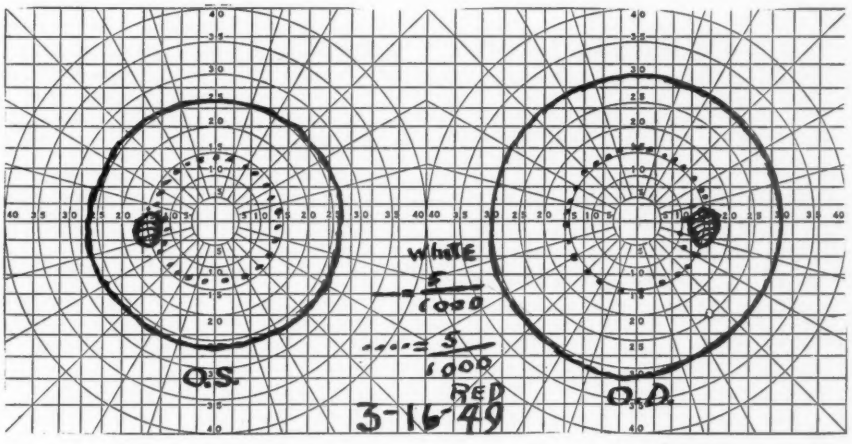

Fig. 2. (Raiford) Visual fields at 3-16-49. There is improvement in the white color fields. The blind spots are normal in size.

The *chief complaint* was that of poor vision in both eyes which was first noted in December, 1947. He had no history of any intercurrent disease, either opthalmological or systemic other than a fall which resulted in a contusion of the forehead without any noticeable effects in October. When returning to school after the Christmas vacation he noticed that he was unable to see the blackboard. There was no pain or redness nor any other symptoms. He was hospitalized from the middle of February to mid-April. The diagnosis at that time was optic neuritis, having had spinal fluid examinations, six typhoid injections and multiple vitamins. These studies were repeated in another hospital and confirmed. The patient's visual defect gradually increased, however, up to the time of hospitalization (mother's statement) of June of this year.

Family History: Parents have been in excellent health. One sister, normal.

SYSTEMIC REVIEW

Ent.: Tonsils had been removed at five years of age. The patient had complained of transitory frontal headaches that were noticeable by his mother in November and December, 1947. They were dull and diffuse in character. He has suffered with no headaches since that period.

Neck: Negative.

Heart and Lungs: Negative.

Abdomen: Appetite was good and bowel function normal. No history of nausea or vomiting.

Genito-Urinary System: Negative.

Extremities: Negative.

Skin: Negative.

PHYSICAL EXAMINATION

The patient had no physical abnormalities other than that of ophthalmologic defects.

Eyes: There was no exophthalmos, ptosis or nystagmus. Pupils were round, central, reacted equally to light, accommodation and were consensual.

Tension: OD 17. OS 19.

Extra-Ocular movements were normal and full.

Lids and Lacrimal apparatus were normal.

Vision: OD 2/60. OS 2/60.

Refraction under atropine cycloplegic 6-17-48:

OD + .50 sph. = o cyl. = 2/60 pinhole 2/60
OS + .50 sph. = o cyl. = 2/60 pinhole 2/60

The patient complained of his vision being smoky.

Visual Fields: 6-18-48 (Fig. 1). There was concentric contraction of the central fields with slight enlargement of the blind spots. No scotoma was present. The patient could not see less than 5 mm. white test object clearly.

Sclera and Cornea: Normal.

Lenses: Were clear and normal.

Vitreous: Clear.

Fundi: The discs were pale bilaterally and both outlines were hazy. A binocular ophthalmoscopic examination was made and no evidence was present that suggested old papilledema. The blood vessels were normal with an arteriovenous ratio of 2 to 3. The macular area revealed slight pigmentation. The periphery was clear. No hemorrhages or exudates were present.

A neurological examination by Dr. Joseph Yaskin did not reveal any other abnormalities.

On June 18, 1948, x-rays were taken of the skull, orbits, sella and optic foramen and were negative. A ventriculo-encephalogram was done June 29, 1948, by Dr. Finkelstein and the following reported. "Only a small portion of the third ventricle is visualized and this is normal in size. The fourth ventricle was not visualized. No air can be seen in the basal cisterna or the subarachnoid pathways. We are, therefore, unable to exclude the presence of a posterior fossa lesion, such as a tumor or basal arachnoiditis."

In view of these combined findings a right frontoparietal craniotomy was done by Dr. Robert A. Groff on July 6, 1948, and a chronic arachnoiditis was found around the optic nerves and chiasma. There fibrous bands and adhesions were incised and the optic nerves freed.

The patient made an uneventful recovery and was discharged on July 13, 1948.

The patient was last seen on March 16, 1949 at which time the visual acuity had improved to OD 6/22.5, OS 6/7.5. The visual fields demonstrated considerable enlargement with the red field being within normal limits. Both fundi revealed some pallor of the discs and otherwise were normal. The patient had attended school throughout the year with the attainment of a high scholastic record.

SUMMARY

A case of optochiasmatic arachnoiditis in an eight-year-old male is reported. The condition was diagnosed from the history, clinical findings, and roentgenologic studies. Surgical exposure confirmed the diagnosis and following the liberation of the arachnoid adhesions about the chiasma the patient showed marked improvement of his visual acuity. The significant fact is that this is the youngest patient reported in the literature confirmed by surgery.

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THE TREATMENT OF BACTERIAL ENDOCARDITIS: REVIEW OF TWELVE CASES

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Wagner[1] has summarized the present status of the treatment of bacterial endocarditis from 521 cases reported prior to January, 1948. It seems clear that a knowledge of the organism present in the individual case is of primary importance. As emphasized by Oglesby[2] and others, there is little need from the therapeutic standpoint for the terms "acute" and "subacute".

We are presenting the experience with twelve cases observed at the Medical College of Virginia since 1944 when penicillin first became available.‡

General study was done on all patients. This included frequent urinalyses, blood counts and blood cultures; sedimentation rates; agglutination tests for typhoid, paratyphoid, brucellosis, tularemia, and against B. proteus X-19; various blood chemical determinations in different cases; electrocardiograms; and x-rays of the heart and lungs.

Prior to April, 1947, blood cultures were done by putting 5-10 cc. of blood in 50 cc. of veal infusion broth-agar and incubating at 37° C. Since that time the same amount of blood has been placed in 50 cc. of tryptose phosphate broth containing 50 mgm. of para-amino-benzoic acid. Gram stains were made every second day during the first week and at the end of the second week. If organisms were seen, sub-cultures were planted on blood agar plates. The colony characteristics on these plates determined the classification of the organism into the alpha, beta, and gamma groups. In one case further sub-classification was done by cultural methods.

The sensitivity of the organism to penicillin was determined as follows: Penicillin was diluted with saline and poured in blood agar plates so that they contained concentrations ranging from 50 to .0375 units per cc. From the original blood agar plate on which the classification was made the organisms were inoculated into blood broth and incubated for twenty-four hours. This culture was diluted 1-1000 with nutrient broth and planted on the above prepared penicillin blood agar plates. The sensitivity was considered to be between the concentration on which growth occurred and the next more concentrated where no growth occurred; thus, if growth was free on the plate containing 2.5 units per cc. and poor or none on that containing 5 units per cc., the report read "sensitive to between 2.5 and 5 units of penicillin per cc."

Blood levels of penicillin were obtained thus: Concentrations of the drug from 10 to .02 units per cc. were made. A twenty-four hour broth culture of a sensitive staphylococcus was diluted 1-1000 with plain broth, mixed with warm agar, and poured on plates. Small cylinders were placed on these plates and the dilutions of penicillin poured into them. The unknown serum was placed in similar cylinders on similarly prepared plates. When the inhibition of growth by the patient's serum was the same as that of a known penicillin dilution, the serum was reported as containing that many units per cc. This indirect method obviously does not determine the patient's penicillin activity against the organism causing his disease and it may well be that the procedure suggested by Schlicter and MacLean[3] will furnish more useful information.

The essential data are presented in the chart.

SUMMARY

In five instances of endocarditis caused by alpha hemolytic streptococci in patients presumed to have rheumatic heart disease, sterility of the blood was readily obtained with penicillin. In four cases the drug was given intramuscularly every two or three hours. In one (Case 2) continuous intramuscular drip was employed but had to be discontinued because of local tissue reaction; thereafter

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CASE	Hospital Number and Date of Admission	Sex, Age, Race	Duration of Disease in Days	Preliminary Incident	Heart Lesion	Type of Organism	In Vitro Sensitivity	Daily Dose of Penicillin in Units	Days on Penicillin	Total Units	Blood Level Obtained Units Per cc	Clinical Course and Complications	Results
1 (L. S.)	A-47713 12/ 2/44	M 40 C	30	Extraction of teeth "Bad cold"	R H D Mitral and aortic	Alpha hemolytic strep.	Not done	160,000	4	550,000	Not done	*1 Clinical improvement, blood cultures positive. No more drug available.	Clinical evidence of renal and pulmonary infarction. Temperature to 101° F., at times, for no reason. Progressive heart failure with death six months after apparent recovery from infection. Blood cultures remained negative after third course. No autopsy permitted.
								120,000	5	575,000	Not done	Again improved, blood cultures positive. No more drug available.	
								320,000	6		Not done		
								240,000	38	11,000,000+	Not done	Marked improvement, blood cultures negative. *2	
2 (D. F.)	B-7926 6/17/46	F 54 W	35	None	R H D Mitral insufficiency Mitral stenosis?	Alpha hemolytic strep. (Salivarius)	0.015	400,000–500,000 *3	40	20,000,000+	Not done	Temperature normal on 6th day. Blood cultures became negative and remained so. Peripheral and cerebral emboli suggested after bacteriologic cure.	Died suddenly while talking to friends 90 days after treatment begun. No autopsy.
3 (D. T.)	B-21173 3/ 9/47	F 60 W	75	None	R H D? Mitral insufficiency?	Alpha hemolytic strep.	0.15–0.30	600,000	35	20,000,000+	0.1–0.5	Temperature normal in 24 hours. General improvement prompt. Petechiae periodically for 11 days after blood was sterile.	Well 15 months after treatment begun.
4 (R. K.)	B-24258 5/28/47	F 16 C	120	Tooth extraction "Bad cold"?	R H D Mitral insufficiency	Alpha hemolytic strep.	25.0–50.0	640,000 2,400,000	3 41	60,000,000+	Not done	Temperature normal on 5th day. During latter part of treatment there were several 2-3 day rises to 100.4° for no apparent reason.	Well 12 months after treatment begun.
5 (C. C.)	B-25853 12/15/47	M 28 C	14	None	R H D Mitral and aortic	Alpha hemolytic strep.	0.075–0.15	640,000	42	26,000,000+	0.5, 0.05, 0.2 *4	Temperature normal on 8th day; blood culture negative on 10th day. Occasional temperature to 99.6° over 1 month after blood became sterile.	Well 6 months after treatment begun.
6 (G. B. S.)	B-25628 5/15/47	F 36 W	60	Tooth extraction	Patent ductus arteriosus	Alpha hemolytic strep.	Not done	150,000–400,000	10	3,000,000+	Not done	Afebrile throughout hospital stay. Blood cultures became negative.	Relapse in 26 days.
						Alpha hemolytic strep.	Not done	300,000–600,000	28	12,000,000+	Not done	Again became positive 26 days after treatment was stopped. Again afebrile. Blood promptly became sterile and remained so. Ductus ligated under penicillin protection 31 days after blood showed no organisms. *8	Well 12 months since blood culture last positive, and 10 months since ductus ligated.
7 (E. C.)	B-30079 7/23/47	M 17 W	270	"Bad cold"	Patent ductus arteriosus and other indeterminate lesion	Alpha hemolytic strep	0.6–1.2	600,000–400,000	32	13,000,000+	0.5–1.0	Temperature exceeded 99° twice, once before treatment begun and again for 4 days, 2 weeks after blood had become sterile while still on penicillin. Ductus ligated under penicillin protection 88 days after blood culture negative. *8	Well 10 months after treatment begun and 9 months after ductus was ligated.

8	(O. L. O.)	A-66360 1/26/46	M 61 W	365 (?)	None	*5	Non-hemolytic strep.	5.0	200,000 320,000 600,000 960,000 *6	2 20 4 43	50,000,000+	0.32	Gradually downhill, with mental disturbance and dyspnea. Blood cultures always positive.	Died on 70th hospital day. Autopsy showed acute bacterial endocarditis, mitral valve, right auricle, and aortic valve with perforation; pulmonary congestion and edema; septic infarct of spleen containing cocci.
9	(N. E. B.)	B-30629 11/24/47	M 55 W	90	None	Patent ventricular septum	Non-hemolytic strep. Microaerophilic	25.0-50.0	1,200,000	52	60,000,000+	0.2-0.5 *7	Temperature normal in 48 hours. Blood cultures sterile thereafter. Temperature to 101° for 4 days, 3 weeks after blood was sterile. *9	Well 7 months after treatment begun.
10	(C. G.)	B-13104 9/16/46	M 25 C	30	Injury to foot	R H D Mitral stenosis	Not recovered		600,000	6	3,600,000+	Not done	Rapidly downhill, dyspnea, orthopnea, and findings indicative of cerebral embolism.	Died on 9th hospital day. Autopsy (brain not permitted) showed mitral stenosis with subacute bacterial endocarditis, mitral valve, aortic valve, and left atrium; cocci present; pulmonary edema; focal embolic nephritis superimposed on chronic pyelonephritis; and splenic infarction (aseptic).
11	(B. B. C.)	B-12729 12/28/46	F 10 W	90(?)	None	R H D Mitral stenosis	Not recovered		800,000	96	76,000,000+	Not done	Gradual general improvement with normal temperature and gain in weight but continued embolic phenomena. Massive cerebral embolus on 94th hospital day.	Died on 96th hospital day. Autopsy showed chronic mitral valvulitis (probably rheumatic fever); subacute bacterial endocarditis, mitral valve, gram positive cocci present; heart's blood culture negative: extensive fibrosis of myocardium; infarcts of spleen, kidneys, and brain; and chronic passive congestion and edema of lungs.
12	(L. S.)	B-17631 12/ 1/46	M 46 W	42	None	R H D? Mitral and aortic	Not recovered		360,000 720,000 1,200,000	9 10 15	28,000,000+	Not done	Transient early improvement with later recurrence of sepsis, pulmonary infarction? and cardiac failure.	Died on 71st day after admission. No autopsy obtained.

*1—Patient received sulfadiazine, 6 grams daily, intermittently.
*2—Received heparin for four days.
*3—Received 500,000 units daily as continuous intramuscular drip for 22 days, resulting in indurated site with temperature 102°; intermittent intramuscular injection used thereafter.
*4—Received caronamide, 4.0 grams every four hours, practically throughout treatment. Effect on blood level not determined.
*5—Aortic valve lesion—See autopsy report.
*6—Received benzoic acid, 2.5 grams T.I.D., and sulfadiazine, 6.0 grams daily.
*7—Level rose from 0.2 to 0.5 after seven doses of caronamide, 2.0 grams every three hours.
*8—Operation by Dr. I. A. Bigger.
*9—Nineteen teeth extracted in four sittings one month after blood became sterile. Socket cultures showed N. catarrhalis, alpha hemolytic streptococci, and E. coli.

intermittent intramuscular injection was used. In one patient (Case 1) clinical but not bacteriological recovery was obtained twice with small doses; subsequently, larger and more protracted dosage resulted in sterility of the blood; however, this patient progressively declined and died from cardiac failure six months after treatment. In three patients (Cases 1, 2 and 3) petechiae and/or embolic phenomena were noted several days to weeks after the blood cultures showed no growth, and in one of these (Case 2) the manner of death suggested embolism (no autopsy permitted). In Cases 1 and 4 there were short periods of inexplicable elevation of temperature after recovery seemed to have taken place. Blood levels of penicillin were obtained in two cases. In one (Case 3) the level equalled that to which the organism was sensitive *in vitro;* in a second (Case 5) the level was five times greater than the sensitivity in the laboratory. In Case 4 the sensitivity test suggested the need for 25 to 50 units of penicillin per cc. Although no levels were done in this case, experience in others made it unlikely that any such levels were reached with the 300,000 units q. 3 h. used. In all three recovery was prompt and has persisted.

There were two cases of endocarditis due to alpha hemolytic streptococci in patients with patency of the ductus arteriosus. In one (Case 6) temporary recovery was obtained with intermittent intramuscular penicillin, followed by relapse in twenty-six days with clinical and bacteriological recovery after retreatment with larger and more protracted dosage. Before the elapse of sufficient time for the evaluation of this second course the congenital defect was repaired and the patient has been well for ten months; in the other recovery was prompt and has persisted for ten months after treatment was begun and nine months after the ductus was ligated, despite the probable presence of some other defect or defects. This second patient had an inexplicably elevated temperature for four days, thirteen days after the penicillin had been started and more than a week after the blood had become sterile.

There were two cases of endocarditis due to nonhemolytic streptococci. Recovery was prompt in one (Case 9) although the blood levels appearing necessary from laboratory study were not approximated. The level obtained appeared to be doubled following the administration of caronamide[4]. In the other (Case 8)‡ there was a gradual downhill course with death from sepsis and cardiac failure. Here also the blood levels obtained were less than those indicated as necessary by *in vitro* testing. However, the discrepancy was not as great as in Case 9, who recovered.

In three cases of endocarditis where the clinical course made this diagnosis certain (two proven at autopsy) but no organisms were obtained, there were no recoveries. One (Case 10) died from heart and kidney failure plus a possible cerebral embolus before therapy could be adequately tried; a second (Case 11) improved and appeared to be recovering, only to die from a cerebral embolus after she had seemed well for three weeks; and the third (Case 12), after temporary clinical improvement, succumbed to sepsis, heart failure, and possible embolism.

General Observations

1. Sterility of the blood and clinical recovery was obtained in endocarditis due to the alpha hemolytic streptococcus by the injection of penicillin intramuscularly at 2 to 3 hour intervals. When relapses occurred, there was subsequent response to larger and more protracted dosage by the same route.

2. The clinical results did not run parallel with the *in vitro* determinations of sensitivity and blood levels by the methods used.

3. Despite recovery from the infection, death ensued from progressive heart failure in one case.

4. Petechiae and/or embolic phenomena occurred after apparent clinical and bacteriological cure. These did not necessarily have any bearing on the ultimately good result; on the other hand, death appeared to result from such an embolus in one case.

5. Elevations of temperature took place after the blood had become sterile. They appeared to be of no significance.

6. Death occurred in the three patients from whose blood no organism was recovered.

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‡A detailed report on this patient by Dr. R. C. Cecil will appear in the *American Heart Journal.*

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William Potts Dewees was the first great American obstetrician. His *Compendious System* of *Midwifery* went through twelve editions. It was largely instrumental in turning American obstetrics from slavishly following the English school to the French school—the abandonment of the lateral obstetrical position for the dorsal. Thoms, writing in 1933, is surprised at "the small amount of real progress that has been made in obstetrical science since his time."

Dewees was of Swedish descent. He was apprenticed to Dr. William Smith of Philadelphia and graduated B.M. at the U. of Penn. in 1789. He began practicing in the village of Abington, just outside of Philadelphia. It was here in September 1789 that he first noticed the effect of bleeding on the course of difficult labor. This patient had a history of very difficult labors, many of her babies being delivered with the crotchet. At the onset of the present labor she had a severe hemorrhage from the lungs and to everyone's astonishment she had a quick easy labor. He reasoned "it could be from no other cause than relaxation, produced by the excessive and alarming haemorrhagy. I quickly resolved to take advantage of this kind hint, by endeavoring to imitate this good example." His subsequent experience in Abington confirmed his surmise and when in 1793 after the great yellow fever epidemic, he returned to Philadelphia to practice and to get his M.D. degree, he chose as his graduation thesis, *The means of lessening pain, and faciliating certain cases of difficult parturition.* (95 pp. 1806). This gained for him so great a reputation that he became a candidate for the newly created chair of midwifery which was established in the University of Pennsylvania in 1810.

XUM

THE TREATMENT OF POST THROMBOPHLEBITIC SEQUELAE IN THE LOWER EXTREMITIES*

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Thrombophlebitis refers to a partial or complete occlusion of a vein by a thrombus accompanied by an inflammatory reaction in the wall of the vein. Serious sequelae follow thrombophlebitis of a large vein, such as the ilio-femoral, and this paper is concerned with a discussion of these sequelae and their treatment. It is necessary to understand the pathology of the acute thrombophlebitic episode and the subsequent course of events in order to appreciate the syndromes that may follow.

During the first twenty-four to forty-eight hours after thrombophlebitis is initiated, the thrombus occludes the vein and return of blood through the vein is impossible. Increased venous pressure occurs peripheral to the obstruction and capillary pressure is so increased that osmosis is in the direction of the tissue spaces. Capillary anoxemia is likewise present and resorption of tissue fluid and crystalloids is inhibited. The associated venitis and perivenitis initiated impulses which irritate the lumbar sympathetics and vasoconstrictor spasm occurs in the peripheral arterioles. Capillary anoxemia is further aggravated and edema increased. Lympathic channels caught in the perivenous inflammation become obstructed. Increased lymphatic pressure results in dilatation of the lymphatic capillaries. The lymphatic system is thus prevented from performing its normal role of removing tissue fluid and proteins; and lymph in dilated lymphatics increases the leg swelling even more.

As thus envisioned, three factors—venous obstruction, arteriolar spasm, lymph stasis—contribute to the leg swelling of acute ilio-femoral thrombophlebitis or milk leg. During the acute venous obstruction, venous return is maintained through the collateral venous channels, mainly through the long saphenous and its tributaries. Usually within a few days dilatation of these superficial veins is apparent. Venous pressure in the saphenous system is increased threefold as the result of its overload.

The above described state of affairs rarely persists over a few days. Soon clot retraction occurs and some restoration of the lumen of the femoral vein results. Pressure in the veins of the leg distal to the obstruction gradually decreases. Resolution of the thrombus begins within a few weeks and soon the femoral system is more or less patent again. But the involved segment rarely returns to normal. The vein walls remain thickened, the valves damaged and incompetent. Blood flows again through the femoral system, but the rate is retarded and chronic venous insufficiency results. The saphenous system is relieved of its overload, but the once dilated veins fail to resume their normal size and tone, and varicose veins result. Subsidence of the inflammatory reaction in the perivenous tissues relieves the lymphatic traffic block in the groin and drainage through the lymph channels again occurs with further reduction in the leg edema.

Arteriolar spasm likewise usually disappears although occasionally it persists for long periods of time, producing a causalgic-like state, as later described. Recanalization of the thrombus, resulting in restoration of venous flow, probably occurs in 95% of cases of ilio-femoral thrombophlebitis. However, permanent damage to the venous and lymphatic system becomes evident as soon as the patient becomes ambulatory. Only the orthostatic factor is needed to bring out the telltale signs of a previous episode of ilio-femoral thrombophlebitis—leg edema from foot to groin with a characteristic "thick" feeling in the thigh. Should the edema be uncontrolled, vigorous growth of fibroblasts occurs in the protein rich tissue fluid. This results in further obstruction of the circulatory channels, especially lymphatic, with ever increasing edema. Varicosities appear in the damaged saphenous system. Fibrous tissue invasion of the subcutaneous tissues further hypertrophies the extremities. Years later fibrous contraction results in indurated cellulitis, the formerly swollen parts now being quite shrunken and woody hard. Nutrition of the covering skin is impaired due to the chronic venous insufficiency, and anoxemia. Eczema, dermatitis, epidermophytosis, episodes of lymphangitis streptogenes, and indolent leg ulcers begin to complicate the picture.

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The sequelae of an attack of acute ilio-femoral thrombophlebitis will depend on the extent of the initial pathology and the adequacy of the treatment of the acute episode plus the efficiency with which the subsequent edema tendency is controlled. We recognize the following post thrombophlebitic states:

1. Chronic venous insufficiency;
2. A causalgia-like state;
3. Chronic hypertrophy and edema;
4. Varicosities of the saphenous system secondary to deep vein thrombophlebitis;
5. Recurring lymphangitis streptogenes;
6. Recurring deep vein thrombophlebitis;
7. Chronic indurative cellulitis;
8. Indolent leg ulcer.

Chronic Venous Insufficiency. This is evidenced by very slight bluish coloration of the skin and mild orthostatic edema. The leg appears entirely normal after a few hours of recumbency. A novocaine block of the lumbar sympathetic ganglia performed even several weeks or months after the acute attack may be remarkably effective in improving the leg. Or a thin elastic stocking is sufficient to control the edema. The role of spasm in producing the edema may be determined by an Ochsner test. The patient is asked to walk a given distance—say a mile—and then measurements are made of the circumference of the affected limb. A novocaine paravertebral block of the related lumbar sympathetics is then performed. After waiting about thirty minutes to obtain full vasodilatation, the patient is asked to walk again a mile and the limb measurements are taken thereafter. A reduction in measurements indicates a considerable spastic element in the causation of the edema and repeated novocaine blocks or a lumbar sympathectomy are in order.

Causalgia-Like State. In this syndrome the gross sequelae of the acute ilio-femoral thrombophlebitis seem minimal, but the patient complains of severe pain in the extremity. The pain complex becomes firmly established early in convalescence. There is usually a stocking-like paraesthesia and evidence of vasomotor disturbance—sweating and coldness of the extremity. Presumably the diseased femoral or iliac vein has continued to over-stimulate the sympathetic system, prolonging the peripheral vasospasm, initiated by the acute episode. Again paravertebral novocaine injections of the sympathetics may be entirely curative. Or a lumbar sympathetic ganglionectomy may be in order.

Chronic Hypertrophy and Edema. (Elephantiasis). The extremity is enormous from foot to groin—a condition called elephantiasis. Orthostatic edema aggravates the picture, but the enlargement is present even after bed rest. The condition results from failure to control completely the edema of the acute and convalescence stage. The hypertrophy is due to fibroblastic proliferation in the edematous tissue and is permanent. The only effective treatment is surgical removal of the affected soft parts—subcutaneous tissue and fascia—by a Kondoleon procedure. Recent modifications of this operation are described by Pratt and Wright and Vots, Barnes and Pratt. Unless repeated attacks of lymphangitis streptogenes, local thrombophlebitis, or epidermophytosis have complicated the picture, the skin in these cases is relatively normal. Primary wound healing may be expected after most extensive procedures. This is to be contrasted with chronic indurative cellulitis, subsequently to be described, in which skin grafting must often supplement the Kondoleon procedure because of the presence of eczema, ulceration, devitalized skin.

Varicosities of the Saphenous System Secondary to Ilio-Femoral Thrombophlebitis. Reference has been made to venous return by way of the saphenous system during the acute deep vein obstruction. This frequently results in newly formed varicosities or aggravation of a pre-existing varicose state. Peculiar as it may seem, permanent dilatation of the saphenous system, that is to say, varicose veins, may constitute the most serious sequela of the deep vein disease. Thanks to modern clarification of the post thrombophlebitic state, we can usually eradicate these varicosities by methods now employed in the non-phlebitic leg. For years a past history of "milk leg" was listed as a contra-indication to saphenous system surgery because of the belief that the deep vein system has been permanently occluded and that the superficial system is needed for venous return, no matter how varicosed. We now know that the affected deep veins recanalize within a few months in the great majority of cases. Contrary to performing a useful purpose, the varicosities actually increase the chronic venous insufficiency and should be eradicated. Our present plan is to wait approximately a year after the acute attack and perform a high saphenous ligation, or high ligation and stripping, followed by sclerother-

XUM

apy of any remaining varicosities. Tests are always performed, of course, to detect the occasional case in which the deep vein has remained occluded, contraindicating saphenous ligation. Persistent occlusion of the deep venous channels may be suspected if Perthes' test is positive, if a tight elastic support aggravates the patient's symptoms, if there is evidence of advanced deep vein insufficiency, as indicated by extensive collateral varicosities across the lower abdomen. Occasionally study by venography may be needed to clarify the situation.

Recurring Lymphangitis Streptogenes. An edematous leg is particularly susceptible to bacterial invasion. The patient who has had ilio-femoral thrombophlebitis and disregards the subsequent leg edema is inviting infection. Epidermophytosis, eczema, and ulceration will render the leg even more vulnerable. The streptococcus is the usual offending organism and a typical attack may be described as follows: The chronically edematous leg suddenly becomes much more swollen. The involved skin, usually of the lower leg only, becomes hot, raised and fiery red. The skin appearance is so akin to that of erysipelas that the term "erysipeloid" has been applied to it. Constitutional symptoms—high fever, chills, prostration—are present. With or without therapy the attacks usually subside in five to ten days, but therapy can greatly shorten the attack and lessen the permanent damage that would otherwise occur, for the pathology is a streptococcal infection of the superficial and deep lymphatics of the skin and subcutaneous tissue, and each attack damages more lymphatics, leaving the limb more edematous and more susceptible to further episodes.

It is, therefore, important to institute treatment promptly and vigorously. Our treatment is as follows: We make an initial painting of the involved skin with 20% silver nitrate solution, followed by an application of voluminous wet dressings of the same solution in dilution of 1 to 20,000, the extremity being highly elevated on an inclined plane, and large doses of penicillin are given parenterally. Treatment is not discontinued until every vestige of edema and infection is eradicated. We discharge the patient with a careful explanation of the invariable recurrence unless a strong elastic support is worn and the extremity kept scrupulously clean. Sulfadiazine, grains fifteen, four times daily for a week of each month, or penicillin therapy for a like period may be necessary to prevent further attacks. Desenex powder or ointment is prescribed for cases initiated by epidermophytosis.

Recurring Deep Vein Thrombophlebitis. Occasionally a patient who has had ilio-femoral thrombophlebitis will be subject to recurring episodes. Attacks more or less similar to the original one recur from time to time. This may be coincident with a possible cause such as an operation, childbirth, or a debilitating disease. Or the attacks may come on spontaneously while the patient is ambulatory and leading a normal life. The extremity swells from foot to groin, tenderness is present along the course of the femoral vein and constitutional symptoms occur. The patient usually has to be treated as she was in the initial attack, with large warm compresses, leg elevation, lumbar sympathetic novocaine blocks and anti-coagulent therapy. We feel that these cases represent instances of an abnormal clotting tendency or severe damage to the wall and valves of the femoral system and believe that superficial femoral vein ligation should be performed at the beginning of the recurring attack or after it has subsided. Only in this way can further episodes be prevented, and, more important, the inevitable chronic indurative cellulitis, next to be discussed, be prevented.

Chronic Indurated Cellulitis. This is the late result of neglected thrombophlebitic edema. Most commonly it is a sequela of ilio-femoral thrombophlebitis, but an identical condition can develop from repeated episodes of local thrombophlebitis in varicose veins. Years of uncontrolled leg swelling have permitted extensive growth of fibroblastic tissue which now has contracted, as is the wont of all scar tissue, to produce permanent induration and fibrosis. Attacks of lymphangitis streptogenes or episodes of thrombophlebitis in local varicosities have contributed to the damage. Such a leg presents a characteristic appearance. The tissues, formerly the most edematous, are now the most contracted, so that the lower leg appears quite narrow as contrasted with the hypertrophied calf and ankle. The skin is discolored and shining and the subcutaneous tissue feels woody hard. Very little pitting edema is now present. Here and there a venous sinus may be found, but most varicosities have been obliterated by previous thrombosis. Scars of old ulcers may be seen, or an indolent leg ulcer may be present.

We feel that conservative therapy has little to offer these patients. A period of bed rest may tide over the attack of dermatitis or cellulitis, or heal an early ulcer. Unna paste boots or elastoplast bandages worn for many weeks may temporarily reduce the leg swelling, and add comfort to the patient. Large indolent ulcers may be similarly made to heal but the healing is rarely long lived, for irreversible unalterable structural changes exist in the skin and soft parts. Chronic venous insufficiency, anoxia, fibrosis, destruction of lymphatics, thrombosed veins, calcified fat and fascia form a formidable pathological background. The congested skin rests upon fibrosed subcutaneous tissue which is in turn compressed between the hidebound skin and a stonelike deep fascia. One hardly could expect to restore such a leg to normalcy by any form of treatment, for it is the result of years of neglect. However, no matter how extensive the skin, the subcutaneous tissue, and the fascial damage is, the structures beyond the fascia are invariably remarkably normal. Muscles are red, well vascularized and non-edematous. Tendons and bones are uninvolved. These facts have led me to employ a modified Kondoleon operation for these cases of chronic indurated cellulitis.

The procedure consists essentially of removing all damaged tissue, skin, subcutaneous fat and fascia, and placing normal skin on normal muscles with no intervening subcutaneous bed or fascia. The steps in the operation are as follows: a longitudinal incision is made through the center of the indurated tissue. This usually involves an incision from the internal malleolus to a point several inches below the inner aspect of the knee joint. No tourniquet is applied. The incision is rapidly deepened through the fibrosed calcified subcutaneous tissue and through the deep fascia until the sub-fascial space gaps wide open. Normal muscles and tendons are exposed. The full thickness flaps of skin, subcutaneous tissue and fascia are now readily stripped back to the tibial crest anteriorly and the mid-line posteriorly. Perforating veins, usually incompetent communicators, are clamped and divided. The flaps are now turned out and the deep fascia is incised from within out, the whole length of each flap, thus delineating the wedge of tissue on each side to be removed. Allis clamps are now applied to the skin edges and Ochsner's to the fibrosed subcutaneous tissue and each flap pared of all subcutaneous tissue and fascia. If the long saphenous has not by now been removed it is now stripped. All bleeders are clamped and coagulated with the Bovie apparatus. A few stab wounds are made near the edge of each flap for drainage. The skin flaps are inspected for viability. Should this seem good, excess skin is simply excised and the skin margins approximated with continuous fine dermal. The flaps are pox-marked to remove tension and small penrose drains are placed through the stab wounds previously made.

Should insufficient normal skin be left for primary closure without tension the skin edges are tacked down to the underlying muscles and a split thickness graft applied to cover the defect. The graft is sutured in place with fine silver wire and covered with xeroform gauze. Flat gauze, moistened in saline solution, is applied to furnish even compression. A sterile catheter is inserted within the dressing, further compression obtained with mechanic's waste or sponge rubber and the leg wrapped firmly with an ace No. 8 leg bandage. A posterior metal splint is applied. The graft is kept moist by injecting glycerine 15% acetic solution ¼% alternating with tyrothricin solution (0.5 mgms. per cc.) through the sterile catheter every eight hours. This prevents pyocyaneus infection and facilitates drainage. The first dressing is usually on the 7th post-operative day. Stitches are not removed until the flaps are well healed, or about the 10th day. An Unna paste boot is often applied before the patient becomes ambulatory.

This operation removes all the diseased tissue—devitalized skin, calcified fat, fibrosed subcutaneous tissue, the contained thrombosed veins, the stagnant venous and lymphatic pools, and the thickened binding fascia. Whereas the cosmetic results are not perfect, the permanent results are good. Subsequent leg edema is minimal and indolent ulcers practically never again develop.

The Indolent Ulcer. The post-thrombophlebitic ulcer represents a breakdown of the devitalized skin and subcutaneous tissue of chronic indurated cellulitis, which we have just described. It differs from a simple varicose ulcer in that the venous congestion is aggravated by incompetency of the deep and communicating veins. The lumen of the femoral vein, once occluded, is again patent; but its valves are damaged. High back pressures are transmitted down the leg through the deep venous sys-

XUM

tem when the patient stands, lifts or coughs. The valves of the communicating veins are unable to withstand these pressures and are likewise incompetent. As varicose veins are usually also present, there is thus a three-fold cause of the venous congestion—back flow in the femoral system, in the communicating veins, and in the varicosed saphenous. Little wonder that the ulcer, embedded in indurated fibrosed tissue and bedeviled by passive congestion plus lymph stasis resists healing or invariably recurs after healing. The surgical approach to the problem is still far from satisfactory, but a better understanding of the pathology involved has improved our results. Homans in 1916 recommended excision of the ulcer, interruption of the communicating veins, and subsequent skin grafting of the ulcer bed. Linton in 1938 described a procedure which interrupted the communicating veins and eradicated the varicosities at the same operation. Buxton and his associates in 1944, and Buxton and Coller in 1945, reported beneficial results from femoral vein interruption in the post-thrombophlebitic state. Homans in 1946 recommended ligation of both the femoral and the saphenous veins when both are found to be diseased. Following through Homan's suggestions, Linton in 1947 recommended superficial femoral vein interruption, and simultaneous saphenous ligation and stripping for the treatment of post-thrombophlebitic sequelae. There were 34 patients in Linton's series. Twenty-one of these had a history of leg ulcer and in twenty of these the ulcer had remained healed two to fourteen months after the surgery. Twenty-four of Linton's cases had pre-operative edema and the edema was improved post-operatively in fourteen. Only one patient in a group of ten without pre-operative edema developed swelling subsequent to the operation.

Linton's procedure is applicable only when the deep venous system can be demonstrated to be incompetent as by the Trendelenburg test or venography. The Trendelenburg test is performed with the tourniquet just below the knee in these cases. Rapid filling of the veins from below indicates backflow in the femoral system and incompetent communicating veins. Linton deferred surgery until the ulcers were temporarily healed by elastoplast bandages, bed rest and so forth. His operation is as follows:

The superficial femoral vein is exposed distal to the profunda, and the saphenous vein at the femoro-saphenous junction. A needle is inserted into the superficial femoral vein and the venous pressure in this vein is recorded. First the superficial femoral vein proximal to the needle, and then the saphenous is temporarily occluded. Should the venous pressure not rise above 30 cm. saline, both the superficial femoral and the saphenous are ligated. If venous pressure rises above 30 cm. saline, either the femoral or the saphenous is interrupted, but not both. The decision as to which vein to interrupt depends on the results of the Trendelenburg test and the venous pressure readings in the superficial femoral when the femoral or the saphenous is compressed. Linton adds stripping out of the entire saphenous system to his procedure, feeling that this is necessary to prevent recurrence of the varicosities and to interrupt indirectly the incompetent communicating veins.

I feel that the Linton operation falls short in one respect—the failure to remove the devitalized skin, indurated subcutaneous tissue, calcified fascia beneath and about the ulcer. The ulcer heals over a bed incapable of maintaining skin nutrition and the ulcer is bound to recur.

Our present practice in the treatment of these post-thrombophlebitic ulcers includes the following steps: 1. Ligation and division of the superficial femoral if the Trendelenburg test indicates incompetency of the deep veins and communicators. 2. Ligation and division of the saphenous and its branches at the femoro-saphenous junction if the long saphenous system is varicosed, and a like procedure in the popliteal space if the short saphenous is varicosed. 3. Eradication of the varicosities down to the upper limits of the area of chronic indurated cellulitis by vein stripping. 4. Excision of the area of chronic indurated cellulitis, as previously described. 5. Wide excision of the ulcer, including removal of the underlying fascia. 6. Simultaneous or deferred split thickness skin grafting.

Our method of excising the ulcer contrasts sharply with the usually described procedure of cutting around the margins of the ulcer, and through the ulcer bed and awaiting the development of a granulation tissue before skin grating. It consists of unequivocal removal of the ulcer and all underlying tissue down to the muscle. An incision is made through the indurated tissue several inches above

the ulcer margins. This incision is deepened to and through the fascia, as described in the operation for chronic indurated cellulitis. The fingers are then slipped under the fascia and the ulcer-containing-tissue elevated off of the muscles. The ulcer and all underlying tissue—fibrosed subcutaneous bed, thickened fascia, and contained damaged blood vessels—are now readily removed by an elliptical incision to either side, encircling the ulcer. If extensive indurated cellulitis is present the primary incision is extended upwards and the diseased tissue pared from each flap. The operation is completed by suturing the upper part of the incision and applying a split thickness graft directly to the muscles now presenting at the former ulcer site. If the ulcer has been badly infected, or if considerable oozing has occurred, the lower portion of the wound is packed and grafting is deferred for several days.

SUMMARY

The post-thrombophlebitic sequelae of the lower extremities are discussed. A fairly satisfactory surgical procedure for a late and morbid sequela—chronic indurated cellulitis—is described. Superficial femoral vein ligation is advocated as a part of the therapy of the post-thrombophlebitic state when the Trendelenburg test indicates incompetency of the deep and communicating veins. A program for the indolent post-thrombophlebitic ulcer is described, which includes femoral and saphenous vein ligation and saphenous vein stripping, local removal of the tissues involved in chronic indurative cellulitis, excision of the ulcer, and skin grafting. A method of excising indolent ulcer which definitely removes all underlying damaged tissues and permits of primary skin grafting on normal muscle is presented.

100 Medical Arts Building.

Antibiotic Drug for Typhoid Fever.

Treatment of typhoid fever with the relatively new antibiotic drug, chloromycetin, is so effective that patients generally are clear of fever three or four days after the drug is first administered, according to an article in the September 10th *Journal of the American Medical Association.*

"Continued experience reveals that fever disappears during the first three or four days of treatment," Drs. Joseph E. Smadel and Charles A. Bailey of the Army Medical Department Research and Graduate School, Washington, D. C., and Dr. Theodore E. Woodward of the University of Maryland, Baltimore, say.

"Our early observations brought out that relapses of typhoid were common in treated patients. In order to eliminate such occurrences, we have prolonged the course of treatment. Analysis of the results obtained in 44 patients with typhoid who received chloramphenicol [chloromycetin] under our observation has indicated a striking relation between the duration of chemotherapy and the incidence of relapses. A clinical relapse occurred in seven of the 13 patients whose initial course of drug was given for eight days or less. None of the members of another group of 19 patients suffered relapses; this group was comparable to the first in essentially all respects, except that treatment was continued for nine to 14 days. A third group consisting of 12 patients, was treated for 14 to 23 days; relapses did not occur among these patients. All patients in the first group who had relapses responded satisfactorily when chloramphenicol treatment was again instituted.

"These results warrant the following conclusions. Chloramphenicol should be administered in adequate amounts for more than eight days to patients acutely ill with typhoid if relapses of the disease are to be avoided. There appears to be little advantage in continuing treatment for more than 14 days."

XUM

PUBLIC HEALTH

L. J. ROPER, M.D.,
State Health Commissioner of Virginia.

The report of the Bureau of Communicable Disease Control of the State Department of Health for September 1949 as compared with the same month in 1948, and for the period of January through September 1949 compared with the same period in 1948, is as follows:

	Sept. 1949	Sept. 1948	Jan.-Sept. 1949	Jan.-Sept. 1948
Typhoid and paratyphoid	15	16	105	109
Diarrhea and Dysentery	971	547	4,889	3,317
Measles	39	67	21,276	6,130
Scarlet fever	58	28	514	639
Diphtheria	14	16	106	159
Poliomyelitis	85	152	246	410
Meningitis	7	6	73	73
Brucellosis	13	9	55	69
Rocky Mountain spotted fever	11	2	97	60
Tularemia	3	4	28	45

CERTAIN EPIDEMIOLOGIC ASPECTS OF POLIOMYELITIS

In an article in the *Texas State Journal of Medicine* for July 1949, Dr. Gaylord Anderson* has so clearly outlined the currently accepted thinking regarding the method of spread of poliomyelitis, that we quote his summary:

"I have pointed out many analogies between the occurrence of demonstrable poliomyelitis and the common infections of childhood which we tend to accept as well understood phenomena. In particular have I drawn comparisons with measles because of a belief that a close analogy exists between the epidemiology of those two diseases. If we were lacking the rash by which each case of measles can be recognized and were forced to base our diagnosis on the cases of pneumonia, otitis, or encephalitis that occur as complications, we should have a vastly different concept of measles than that which currently prevails. There is, I believe, strong evidence to support the belief that poliomyelitis virus infection is as widespread as is measles, that most of us respond well to the infection through the development of satisfactory resistance, but that a few are so constituted physiologically that they respond badly and are paralyzed. It is in the study of these physiologic variants that I believe we shall find our most fertile field of research."

This concept of the common occurrence of poliomyelitis is also advanced by Hart Van Riper, Medical Director of the National Foundation For Infantile Paralysis, who states "it is now clear that the majority of us become infected at some time during life with the virus of poliomyelitis. The clinical disease and the infection are not the same thing. The disease is rather an exceptional result of the infection."

The acceptance of the theory that the virus of poliomyelitis is so widespread that contact for all is sooner or later inevitable, puts it in the category of childhood diseases like mumps, measles and chickenpox and makes most of our present attempts at control seem feeble and inadequate.

The final control of poliomyelitis depends, in our opinion, upon the production of an effective immunizing agent.

*Dr. Anderson is Mayo Professor and Director, School of Public Health, University of Minnesota, and, with Margaret G. Arnstein, author of the text "Communicable Disease Control".

RESOLUTION PASSED BY THE STATE BOARD OF HEALTH ON THE RESIGNATION OR DR. LAWRENCE T. ROYSTER

WHEREAS, Dr. Lawrence T. Royster resigned as a member of the State Board of Health, September 17, 1949,

BE IT RESOLVED: That the Board recognizes his wise counsel, faithful service and sincere interest over a period of thirty-five years, during which period public health has been advanced from a minor to a major responsibility of Government; and further,

That the Board hereby desires to express its appreciation for his warm personal friendship and devotion to his responsibilities and duties as a member; and further,

That a copy of these resolutions be spread on the minutes of the State Board of Health.

September 29, 1949

BOOK ANNOUNCEMENTS

Aesculapius Comes to the Colonies. The Story of the Early Days of Medicine in the Thirteen Original Colonies. By MAURICE BEAR GORDON, M.D. Ventnor Publishers, Inc., Ventnor, N. J. 1949. xiv-560 pages. Illustrated. Cloth. Price $10.00.

This is a well worthwhile treatise on Colonial medicine. The author takes up each colony separately and precedes his medical discussions with a brief story of its civil history. In the general introduction there is an account of Indian medicine, but it does not appear that the doctors in any of the colonies were influenced by this type of medicine. The author has something to say about practically every medical man in the English Colonies. The work has a good index and should prove a valuable reference work to all who are interested in medical history.

The medical man varied a great deal in the 13 Colonies, but seemingly they had one thing in common and that was a tendency to combine the practice of medicine with other employments. In New England most preachers practiced medicine. This was also true in New Jersey and several congregations complained of the size of the medical fees their preachers charged. In New Hampshire many doctors of medicine became judges. The majority of colonial doctors were apprenticed trained. An amusing story is told of Primus, a slave of Alexander Wolcott. Primus accompanied Wolcott in his visits to the sick and helped in the preparations of the medicines. After many years Wolcott set Primus free and the negro immediately set himself up as a doctor and prospered. On one occasion he was called to see a neighbor of Wolcott's and on leaving the house he called on the doctor and told him that he had just seen his neighbor's child, found it to be a very simple case and had told the mother that it was not necessary for her to send so far for a doctor for Dr. Wolcott would have done for the child as well as anyone else.

Benjamin Franklin is included among the Pennsylvania doctors not because he practiced medicine, but because he founded the first hospital in America and because he was interested in all forms of knowledge as was Thomas Jefferson in Virginia. Franklin's keen insight into human nature and his practical common sense is illustrated by his saying "Quacks are the greatest liars in the world—except their patients." John Wesley is included among the Georgia doctors because he wrote his *Primitive Physick* while he was in Georgia. This little work went through twenty-three editions in Wesley's lifetime and nine others after his death. I was interested to learn that John Wesley assisted in the first postmortem examination performed in Georgia and that George Whitfield established the first hospital and the first free clinic in that state.

There are a few small errors that should be corrected in the next edition. On page 458 after describing the first meeting of Washington and Mercer in the expedition against Fort DuQuesne, there is a statement that "It was Mercer who advised Washington to make Virginia his residence." It also says that Mercer's consultation room and apothecary shop were still standing in 1908. The author might say that it is one of the show places in Fredericksburg. On page 48 "Mr. Eise", the professor of anatomy at St. Thomas' should be "Mr. Else". My only real criticism is the bibliography. It serves no useful purpose except as a demonstration of the wide reading of the author. If the reader wishes to pursue a subject further he gets little help from the bibliography. For instance, I would like to know why the disease that killed off the Indians and so made possible an unopposed landing of the Pilgrims in 1620, was considered to be bubonic plague. I would like also to know more about the epidemic of syphilis that spread among the men, women and children of Boston which started from an ulcer of a nursing mother's breast. In the first instance I imagine that John Brown's article on The Epidemics of the Indians of New England in 1616-1620, Johns Hopkins Hospital Bulletin 20, 1909, would be the place to look. In the second case there is no clue whatever.

M. P. R.

XUM

EDITORIALS

Our New President

WALTER CLEVELAND CAUDILL was born in Alleghany County, North Carolina, June 9, 1888. He was educated at Elk Creek High School, Elk Creek, Virginia, and the Appalachian State Teachers College, Boone, North Carolina. In 1913 he was graduated from the Medical College of Virginia. He interned at Lewis-Gale Hospital, Roanoke, and St. Elizabeths Hospital, Richmond, and located in Pearisburg in 1914.

WALTER CLEVELAND CAUDILL, M.D.
President, Medical Society of Virginia

Dr. Caudill served with the Medical Corps of the U. S. Army and was overseas for 18 months; he was discharged as a Captain in July 1919 and returned to Pearisburg where he has since continued his practice. He was married to Miss Mary Ring Cornett, Elk Creek, Virginia, in 1920 and they have one son, Carrel Mayo Caudill, M. D.

Dr. Caudill has had a varied experience in civic and medical affairs of Giles County. One of his important activities was the establishment in Pearisburg of St. Elizabeths Hospital in 1924, which he has since operated. He assisted in the organization of the local Lions Club and is one of its past presidents. He was the first president of the Bank of Giles County and is now its vice-president and one of its directors. Dr. Caudill is a past-president of the Southwestern Virginia Medical Society and is a Fellow of the American College of Surgeons. Although he is best known for his medical work, he is equally distinguished in the political field. He served for 15 years as Chairman of the Democratic Party in Giles County, and is

now a member of the Ninth District Democratic Committee and the State Central Committee. He was elected to the House of Delegates of the State of Virginia in 1936 and again in 1938. In 1940 he became a member of the State Senate and is at present serving in this capacity. He is also Chairman of a Legislative Commission created by the 1948 session of the General Assembly to study Medical Education in the State.

He has played an active role in the organization and plans for a modern new 50-bed hospital, which will be erected in Pearisburg within the next year.

For the past several years, Dr. Caudill has rendered invaluable service to the Medical Society of Virginia as Chairman of its Legislative Committee. During this time the Society has won all its important legislative bills and has gained, in addition, added prestige and understanding for the medical profession.

President-Elect Caudill assumes the duties of his office at a critical time for the private practice of medicine. The time is not far distant when the doctor and the legislator, without bitterness or emotionalism, must attack the problem and find the solution of better medical care for *all* the people. The Society is truly fortunate to secure the leadership of one who has spent his entire life so near his people, who understands their everyday problems and who bears the genuine admiration and respect of his community. He brings to the Society the perspective of a "grass-roots" physician, the acumen of a successful politician and the innate ability to reconcile differences and to "get things done". These attributes should stand the Society well for the forthcoming year.

—J. P. K.

The Consumer Must Decide

The Consumers' Research Bulletin for September carries an excellent editorial on Compulsory Health Insurance from the standpoint of the consumer. It is so good that we were tempted to copy it in full. It is pointed out that the American consumer has long been used to making his own choice as to how his dollars for products and services are to be spent. Paying for medical and hospital care is an expenditure that does not give consumers a sense of pleasure and well-being as does the purchase of a new automobile, a television set or a fine steak. The successors of the O.P.A. ideologists have seized on this fact in their attack on the consumer's method of handling his hospital and doctor's bills. The U. S. proponents of compulsory health insurance have skillfully disguised the compulsion aspect by referring to their program as "National Health Insurance" and their goal is better health for the entire nation.

The problem for the consumer is further complicated by "as shrewd an advertising and promotional campaign as was ever put on by patent medicine manufacturers in their palmiest days." Furthermore it is difficult for a consumer to form a fair idea of what the plan will cost, the quality of medical care he will get or how the plan has worked in other countries. There are eight volumes or nearly 5,000 pages of reports of the hearings before the 79th and 80th Congresses. *The Consumers' Research Bulletin* suggests that the consumer read Mr. Kenneth C. Crain's analysis of the material which was published in *Hospital Management* for January 1939.

The United States Consumers must say whether they want the Federal Government to have the *power* to decide how much of their income *must* be sent to the United States Treasury for medical care. "Can they judge from the sales claims whether the new product will be better than the one they are now using? The

XUM

decision is in their hands, but it will be in the hands of politicians, if consumers do not seriously and promptly, concern themselves with the problem."

British Health Service from a Druggist's Standpoint

The American Druggist devotes its July number to a consideration of the British State Medicine. It is a very attractive issue. The editor, John W. McPherrin, spent five weeks in England studying how the plan is working. He reports that the druggists in England are divided. Those who were doing well before July 5, 1948, are against it and those who were doing poorly are for it. The average price for a prescription in England is now fifty cents as compared to the average United States price of $1.40. The average retailer is receiving 50 per cent more for "government" prescriptions than he did under the old National Health Insurance, but an average of 25 per cent less than he received for private prescriptions. The pricing of prescriptions is done by a central Bureau or Pricing Offices. The druggist sends all prescriptions to this office. He must do them up in two bundles, one for special prescriptions and appliances and for prescriptions whose ingredients cost more than a dollar and the second for the ordinary prescriptions.

Mr. McPherrin interviewed a great number of British customers. The average Briton wanted this program very much. Today he is reluctant to admit that there is anything wrong with the scheme for fear it will reflect upon his own good judgment. In his editorial Mr. McPherrin says that after five weeks in Great Britain he comes back home with a new reverence for the faith of mankind that created America. He says the dictum "The State is my Shepherd, I shall not want" which is the real concept of the Welfare State, has done something to the British spirit.

Bibliotheca Obstetrica

ROSSLIN, EUCHARIUS (d.1526) Der Swangern frawen und hebammē roszgartē. Hagenau, H. Gran. 1513.

The earliest printed textbook for midwives. It survived forty editions, being used as late as 1730. An English translation by Richard Jonas was published in 1540, printed by Thomas Raynalde. This translation, entitled *The Byrth of Mankynde,* was the first book on the subject to be printed in English. For a bibliographical study of the work, *see* Sir D'Arcy Powers' article in *The Library,* 1927. Series 4, viii, 1-37.

The text and pictures are derived from classical times—Soranus and Muscion, as they came down through the Middle Ages in MSS. Gustav Klein in his facsimile edition (Munich, 1910) gives about all that is known of the author. From 1493 to 1498 he was listed as apothecary in the city of Freiburg i.B. and was listed as a house owner until 1526. In 1506 he became city physician of Frankfurt. In 1508 he is known to have visited the Countess Katharine of Braunschweig and Luneburg and it is to her that he dedicated his book. In 1513 he was city physician in Worms but in 1517 he was back in Frankfurt as city physician with a salary of 70 fl. a year. He died in September 1526.

Besides the "byrth figures" the work is noteworthy for reviving Soranus' podalic version. It is also responsible for the myth that Julius Caesar was named Caesar for having been cut from his mother's side.

SOCIETIES

The Southwestern Virginia Medical Society

Held its regular semi-annual meeting October 6, at the Governor Tyler Hotel, Radford, with a good attendance, and several new members were elected. In addition to papers by members, the guest speaker was Dr. Karl J. Karnaky of Houston, Texas, who spoke on Pelvic Endometriosis. There was an attendance at the afternoon session of about thirty-five and at the banquet of 105.

Dr. Harry Hayter of Abingdon was elected president for the coming year; Dr. James P. King of Radford, vice-president; and Dr. A. F. Giesen of Radford, was re-elected secretary-treasurer. The two members elected to the Executive Committee are Dr. D. S. Phlegar of Blacksburg and Dr. Harry W. Bachman of Bristol.

The next meeting will be held in Abingdon in May 1950, the exact date to be named later.

Fairfax County Medical Society.

At a meeting of this Society on September 17th, the following officers were elected: President, Dr. J. D. Zylman; vice-president, Dr. H. Garnett Snead; treasurer, Dr. Emanuel Newman; and re-elected secretary, Dr. Alice H. Kiessling.

Lynchburg Academy of Medicine.

At the regular meeting of the Academy on October 3, Dr. Kenneth L. Pickeral of Duke University School of Medicine gave an interesting talk, illustrated with a movie, on "The Modern Treatment of Burns".

Roanoke Academy of Medicine.

New officers of the Academy, who took office at the October meeting, are: President, Dr. M. A. Johnson, Jr.; vice-presidents, Drs. John Gardner and Churchill Robertson; secretary-treasurer, Dr. W. H. Kaufman. Drs. W. R. Whitman, Sr., and Charles Smith were named as members of the Executive Committee.

The Virginia Peninsula Academy of Medicine

Resumed its monthly meetings in September following the summer recess. The guest speaker was Dr. Julian Ruffin, Clinical Professor of Medicine and Director of the section of Gastro-enterology at Duke University, whose subject was the Value of Laboratory Aids in the Differential Diagnosis of Jaundice. He stressed the fact that every doctor sees an occasional case of jaundice where it is impossible to make an exact diagnosis without the aid of these tests. Failure to make a correct diagnosis will result in a dangerous and unnecessary operation or deny to the patient surgical aid which is badly needed. The speaker gave illustrations of many cases pointing out the value of the laboratory test in each.

The meeting which was preceded by a social hour and dinner was attended by 73 physicians throughout the Peninsula and neighboring counties. Dr. Russell Buxton, president for the coming year presided.

WILLIAM A. READ,
Secretary

NEWS

Afterglow!

The 102d meeting of the Medical Society of Virginia has passed into the halls of memory. We hope that you all enjoyed yourselves. Judging by the way you looked and by what you said, we believe that you were most happy to be at Old Point Comfort.

The management of the Chamberlin Hotel outdid itself in its efforts to please. Your hosts, the Warwick County and the Elizabeth City County Medical Societies, are gratified that all went so smoothly. It has been 69 years since you were last at Old Point Comfort. In the meantime we have gotten out of practice in the art of being hosts to the Medical Society of Virginia.

The only cloud on the festive scene was the enforced absence of our beloved President, Dr. M. Pierce Rucker of Richmond. In July 1947, Dr. Rucker and I were among the American obstetricians who attended the International Congress of Obstetricians and Gynecologists at Dublin, Ireland. This Congress celebrated the 200th anniversary of

XUM

the Rotunda Hospital. I shall always remember the parting words of Dr. O'Donel Browne, one of the secretaries of the Congress and at present the Master of the Rotunda. He said, "Do not wait another 200 years before coming back to Dublin."

On behalf of the medical profession of the Virginia Peninsula, I want to say to the Medical Society of Virginia: Do not wait another 69 years before coming back to Old Point Comfort!

CHESTER D. BRADLEY, M.D.

Another Banner Meeting.

The Warwick and Elizabeth City County Medical Societies, under the chairmanship of Dr. Waverly R. Payne, with Dr. Paul J. Parker as co-chairman, put on such a meeting after sixty-eight years, that it appeared that they were accustomed to staging the State Society meetings every few years. The fact that the Chamberlin at Old Point, with its excellent management, was headquarters, added to the pleasure of those attending. The one thing which kept the meeting from being perfect was the absence of the President, Dr. M. Pierce Rucker of Richmond, who was prevented by personal illness from attending and taking part.

Business sessions were held as usual and there were several guest speakers at the scientific sessions—Dr. Wilburt C. Davison of Durham, N.C., Dr. Mark M. Ravitch of Baltimore, Dr. Arthur T. Hertig of Boston, and Dr. Robert Lomax Wells of Washington. Exhibits—both scientific and technical—were of a high class.

Awards were made the following scientific exhibits for their originality and method of presentation: First, "Lead Glass Fabric. Protective Against Roentgen and Beta Rays" by Drs. V. W. Archer, George Duncan, Jr., H. D. Hebel, John G. Kroll and D. A. Cunningham of the University of Virginia, Charlottesville; Second, "Getting Well in a Mental Hospital" by Department of Mental Hygiene and Hospitals, Eastern State Hospital (Dr. Granville L. Jones), Williamsburg; and Third, "The Effect of Brain Stimulation on Blood Pressure and Kidney Circulation" by Drs. Ebbe Curtis Hoff, Jos. Kell, Jr., Nelson Hastings, Edwin H. Gray, and Dillard M. Sholes, Jr., of Medical College of Virginia.

The House of Delegates selected Dr. Henry W. Decker of Richmond to be nominated to the American Medical Association as the General Practitioner of the Year.

At the general meeting on Monday evening, eighteen physicians became members of the "50-Year Club", the Society thus honoring its members who had been engaged in the practice of medicine for fifty years.

Apart from the boat trip around Hampton Roads, social features were centered in the cocktail party and banquet on Tuesday evening, with Dr. E. L. Alexander as master of ceremonies. Following the banquet, several much appreciated songs were given by the Crusaders Chorus, directed by the professor of music at Hampton Institute. Honorable Joshua W. Lee of Oklahoma was the speaker on this occasion.

Roanoke was selected for the 1950 meeting and the following officers were elected: President-elect, Dr. C. Lydon Harrell of Norfolk; and vice-presidents, Drs. Waverly R. Payne, Newport News; James P. Williams, Richlands; and James W. Love, Alexandria. Dr. Walter P. Adams of Norfolk was elected to succeed Dr. Harrell for his unexpired term on the Council. Dr. W. C. Caudill of Pearisburg succeeded to the presidency.

American Medical Association.

Advance registrations and hotel reservations are now being received for the 1949 Clinical Session—the third annual mid-year meeting of the A.M.A.—to be held in Washington, December 6-9.

Requests for reservations should be made before November 9 and sent to the Chairman of the Subcommittee on Hotels, American Medical Association, Hotel Reservation Bureau, Star Building, Washington 4, D. C.

The Clinical Session will provide a full-scale scientific program specifically designed for the general practitioner. Coordinated with this outstanding scientific program will be approximately 100 scientific exhibits which will present original work on the subjects discussed.

The newest offerings of 125 manufacturing firms will comprise the Technical Exposition. Here will be found the latest developments in scientific medical research, drugs and equipment.

The clinical sessions and the exhibits will be held in the National Guard Amory, Capitol Avenue and East 19th Street. The exhibit hall will be open throughout the meeting, 8:30 a.m. to 6 p.m., giving ample opportunity to study the latest additions to modern medical practice.

Televised surgical and clinical procedures, similar to those shown in color at the A.M.A. annual sessions in Atlantic City last June, will be presented at the Washington meeting. The demonstrations will originate in the Johns Hopkins Hospital and will be shown on screens in the armory.

The House of Delegates will meet at the Hotel Statler during this session. One of the first orders of business will be the annual selection of the general practitioner who has made an exceptional service contribution to his community. A gold medal will go with the honor.

For the convenience of doctors making advance registrations and reservations, The Journal of the American Medical Association is publishing in its advertising section every week, convenient hotel reservation and advance registration blanks. Listed also are the leading hotels and their rates.

Gill Memorial Spring Graduate Course.

The Twenty-Third Annual Spring Graduate Course of the Gill Memorial Eye, Ear and Throat Hospital, Roanoke, will be held April 3-8, 1950. The guest faculty is as follows: Dr. F. W. Stocker, Durham, N. C.; Dr. Conrad Berens, New York City; Dr. Fred Wilson, Chicago; Dr. C. W. Mayo, Rochester; Dr. Edmund B. Spaeth, Philadelphia; Dr. Arthur J. Bedell, Albany, N. Y.; Dr. James J. Regan, Boston; Dr. W. Gayle Crutchfield, Charlottesville; Dr. Stuart Cullen, Iowa City; Dr. Richard G. Scobee, St. Louis; Dr. Daniel S. Cunning, New York City; Dr. Alson E. Braley, New York City; Dr. Karl M. Houser, Philadelphia; Dr. Hugo L. Bair, Rochester; Dr. Gabriel Tucker, Philadelphia; Dr. Edward M. Walzl, Baltimore; Sir Alexander Fleming, London, England; Dr. J. Barrett Brown, St. Louis; Dr. Walter B. Hoover, Boston; and Dr. Oscar C. Hansen-Pruss, Durham, N. C.

Site Given For Richmond Memorial Hospital.

"Laburnum", the home for four generations of the Bryan family has been offered and accepted as the site for the new 300-bed hospital in Richmond, to become part of a living memorial to those Richmonders who lost their lives in the second World War. It consists of fourteen acres and the home on Westwood Avenue, valued in excess of $125,000. It has recently been owned by Mr. and Mrs. David Tennant Bryan, who are giving it as a memorial to his grandfather, Joseph Bryan, and his father, John Stewart Bryan, both of whom had devoted so much of their lives to the betterment of the community. It was built by Mr. Joseph Bryan in 1885 and rebuilt by Mr. John Stewart Bryan after it was destroyed by fire in 1908. Mr. and Mrs. Bryan plan to vacate the property at the convenience of the hospital.

Plans call for a campaign for public subscriptions during the coming winter and additional funds required for the project will be sought under the Hill-Burton Act after the campaign ends.

Dr. Hellebrandt Gets Scholarship.

Dr. Frances Hellebrandt, Medical Director of the Baruch Center of Physical Medicine of the Medical College of Virginia, has been awarded a $750.00 scholarship for specialized study and training in the treatment of cerebral palsy. This scholarship is awarded jointly by Alpha Chi Omega, national woman's fraternity, and the National Society for Crippled Children and Adults. Dr. Hellebrandt has begun scholarship study at the Children's Rehabilitation Institute, Cockeysville, Maryland.

Dr. Morgan Raiford,

Formerly of Franklin, Va. has accepted a position as Clinical and Research Director at the Brady Clay Memorial Eye Clinic, Emory University School of Medicine, Atlanta. For the past three years, he has been at the University of Pennsylvania and in July received his Master of Medicine Science in Ophthalmology.

Winchester Memorial Hospital Staff Conferences.

The following staff conferences are scheduled at the Memorial Hospital, Winchester, and are open to all physicians who desire to attend:

November 16th—The Annual Hunter H. McGuire Lecture—"The Eye in Thyroid Disease" by Dr. John H. Dunnington, Professor of Ophthalmology, College of Physicians and Surgeons, Columbia University, New York City.

January 18th—"Obesity, a Challenge to the Practitioner" by Dr. William Parson, Professor of Medicine, University of Virginia.

February 15th—Dr. George T. Finney, Associate Professor of Surgery, Johns Hopkins Hos-

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pital, School of Medicine, Baltimore. Subject to be announced.

March 15th—"Recent Advances in Otology and the Treatment of Deafness" by Dr. Fletcher D. Woodward, Professor of Otolaryngology, University of Virginia.

April 5th—The Annual Frank N. Hack Lecture —"Diagnosis, Surgery and Outlook in Cancer of the Right Colon" by Dr. C. W. Mayo, Head of Section in Division of Surgery, Mayo Clinic, Rochester.

May 17th—Clinical-Pathological Conference by the Staff of the Hospital.

The Medical College of Virginia,

Beginning its one hundred thirteenth session this year, has the largest enrollment in its history, with 1331 students registered. Of the schools, medicine has the largest enrollment with 354 students, 84 of whom are freshmen and 254 are veterans. There are 27 women enrolled in medicine.

Fracture Meeting.

The Regional Fracture Committee of the American College of Surgeons of Western Pennsylvania will meet at the University Club, Pittsburgh, on November 16th. There will be a conference on fracture problems and Dr. Harold A. Sofield, Chicago, will speak on "Medullary Nailing of Fractures". All members of the profession are invited to this meeting.

Urology Award.

The American Urological Association offers an annual award of $1,000.00 (first prize of $500.00, second prize $300.00 and third prize $200.00) for essays on the result of some clinical or laboratory research in Urology. Competition shall be limited to urologists who have been in such specific practice for not more than five years and to residents in urology in recognized hospitals.

The first prize essay will appear on the program of the forth-coming meeting of the American Urological Association, to be held at the Hotel Statler, Washington, D. C., May 29-June 1, 1950.

For full particulars write the Secretary, Dr. Charles H. de T. Shivers, Boardwalk National Arcade Building, Atlantic City, N. J. Essays must be in his hands before February 20, 1950.

Dr. James Smith Honored.

On September 19th at the Commonwealth Club, Richmond, Drs. Margaret Nolting, Hunter H. McGuire, Douglas Chapman, H. S. Zfass, J. Powell Williams, Kinloch Nelson, H. Joseph Williams, W. T. Thompson, Jr., William H. Harris, Jr., and John P. Lynch, ten of the former students and colleagues of Dr. James H. Smith, gave a birthday dinner in his honor and invited some of his personal friends. Guests included partners in the McGuire Clinic, and Drs. Wyndham B. Blanton and William B. Porter.

Dr. Smith was presented with a silver tray and eight silver cups, inscribed as follows: "Presented to James H. Smith, M.D., in grateful appreciation by those he has trained and inspired."

Dr. J. Powell Williams was toastmaster.

Dr. Smith's response was characteristic and appropriate.

The Division of Alcohol Studies and Rehabilitation

Of the State Department of Health has arranged a one-day symposium on Rehabilitation of Alcoholics to be held on Tuesday, December 6, at the Baruch Auditorium, Medical College of Virginia. There will be morning, afternoon and evening sessions beginning at 9:30 a.m., 2:00 p.m., and 8:00 p.m.

Nationally known authorities have been secured for this symposium, including Dr. Robert Fleming of the Harvard Medical School and Director of the Alcohol Clinic at the Peter Bent Brigham Medical School, Boston, Massachusetts; Dr. Joseph Hirsh, author and member of the Committee on the Problems of Alcohol of the National Research Council; Dr. Giorgio Lolli, Medical Director of the Yale Plan Clinic; Dr. Harvey B. Haag, Dean of the Medical College of Virginia and member of the Division's Advisory Committee; Dr. Raymond G. McCarthy, Executive Director of the Yale Plan on Alcoholism; and an outstanding representative of Alcoholics Anonymous. There will also be a discussion of the Virginia approach to this problem.

The meeting will be opened to the public and is designed to be of particular interest to physicians, educators, public health workers, ministers, and persons concerned with social problems.

State Department of Health News.

Dr. Joseph W. Still has resigned as health of-

ficer for the Frederick-Winchester Health District to enter the field of research. Dr. Paul B. Chapman has been appointed health officer to replace Dr. Still in the Frederick-Winchester Health District.

Dr. Stuart T. Bray has been appointed health officer for Buchanan and Tazewell counties.

Dr. Wilbur R. Southward, Jr., has been appointed Director of the Bureau of Venereal Disease Control of the State Department of Health.

Dr. Edward F. Corson,

A member of the teaching staff at Jefferson Medical College, Philadelphia, since 1920, has been appointed head of the department of dermatology at the college. He has been professor of dermatology since 1947.

Southern Psychiatric Association

The forthcoming meeting of the Southern Psychiatric Association will be held in New Orleans, Monday and Tuesday, November 28-29 at the Roosevelt Hotel. During this meeting papers will be presented by guests and local speakers on topics relevant to neuropsychiatry and allied subjects. An entertainment program will be enjoyed by all attending and this is in the hands of the Committee on Arrangements.

The Medical College of Virginia is somewhat honored by having two of its Alumni holding office during next year's regime, Dr. Walter J. Otis, being President and Dr. R. Finley Gayle, President-elect.

The other officers of the Association are: Dr. R. Burke Suitt, Durham, N. C., Vice President; Dr. Arthur J. Schwenkenberg, Dallas, Texas, Chairman, Board of Regents, with Dr. Joseph L. Knapp, Weston, W. Va., Dr. O. S. Hauk, Nashville, Tenn., members of the Board of Regents; and Dr. Newdigate M. Owensby, Atlanta, Ga., Secretary-Treasurer.

Dr. E. G. Gill,

Roanoke, has recently been made a member of the Omicron Delta Kappa, a national honorary leadership fraternity.

Dr. Darius Flinchum,

Formerly of Stonega, is now on the orthopedic staff of Duke Hospital, Durham, North Carolina. He has a Fellowship from the National Poliomyelitis Foundation.

Dr. G. Everard Godman,

Recently of Chilhowie, is now serving as assistant resident in surgery at the De Paul Hospital, Norfolk. He plans to remain there for at least twelve months.

Post Graduate Medical Instruction at the University of Virginia.

General Practice as a Medical Specialty was discussed by Dr. William L. Pressly, Due West, South Carolina, on September 26th at the University of Virginia. He was the first speaker on the 1949-50 program of graduate medical instruction for house officers and staff members of the University Hospital. Dr. Pressly received the gold medal of the American Medical Association, at its last meeting, for exceptional service by a general practitioner.

Harvard Medical School supplied three doctors as speakers during October. On the 3rd, Dr. Louis K. Diamond spoke on Present and Future Uses of Blood and Blood Derivatives in Therapeutics. On the 24th, Dr. Reginald Fitz presented a birdseye view of Modern Medical Practice, and on the 31st, Dr. Frederick C. Robbins was the speaker.

Van Meter Prize Award.

The American Goiter Association again offers the Van Meter Prize Award of Three Hundred Dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The Award will be made at the annual meeting of the Association which will be held in Houston, Texas, March 9, 10 and 11, 1950, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations; should not exceed three thousand words in length; must be presented in English; and a typewritten double spaced copy in duplicate sent to the Corresponding Secretary, Dr. George C. Shivers, 100 East St. Vrain Street, Colorado Springs, Colorado, not later than January 15, 1950. The committee, who will review the manuscripts, is composed of men well qualified to judge the merits of the competing essays.

A place will be reserved on the program of the annual meeting for presentation of the Prize Award Essay by the author, if it is possible for him to attend. The essay will be published in the annual Proceedings of the Association.

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Richmond Gives Free Tests For Diseases.

Seventy-five thousand Richmonders will be given the opportunity during the next ten months to undergo free tests for seven chronic diseases. This has been made possible by a grant-in-aid from the United States Public Health Service, and the campaign will be conducted by the City Health Department and various local agencies. All who wish it will be given tests for heart disease, diabetes, tuberculosis, obesity, anemia, high blood pressure and syphillis. It will be possible to run 450 tests daily and no individual will be required to spend more than 15 minutes in taking the tests. Results will be forwarded to the family physician for interpretation to the individual.

The Neuropsychiatric Society of Virginia

Is holding its fall meeting at 2:00 P. M. Wednesday, November 2, in Roanoke at the Hotel Roanoke. Dr. James B. Pettis, Superintendent of Western State Hospital, Staunton, is president.

Dr. Harry C. Solomon, Professor of Psychiatry at Harvard Medical School, and Medical Director of Boston Psychopathic Hospital will speak on "Psychiatric Treatment And Its Results." All Psychiatrists and interested physicians are cordially invited to attend Dr. Solomon's lecture.

The Society's professional and business meeting will be concluded by a Social Hour and Dinner that evening at the Hotel Roanoke.

The Council on Industrial Health,

Of the American Medical Association, will hold its tenth annual Congress on Industrial Health at the Roosevelt Hotel in New York City, February 20 and 21.

Spotsylvania Health Board.

This county which has been without an active health board foi about ten years, has arranged to resume this work and the county supervisors have named a board composed of Dr. W. H. Johnson of Spotsylvania and several laymen.

National Conference on Sight.

The National Society for the Prevention of Blindness will hold a five-day conference in conjunction with the Interim Session of the Pan-American Association of Opthalmology, MARCH 26-30, 1950, at the Floridian Hotel, Miami Beach, Florida. The theme of the meeting will be *The Americas Unite to Save Sight,* and among the subjects to be discussed are: Current blindness prevention programs in countries of the Western Hemisphere; Trachoma; Industrial ophthalmology; Eye problems of school children; Medical and social management of the glaucomas.

Persons directly or indirectly concerned with eye health and safety will find this conference of interest. Details concerning the program may be obtained by writing directly to the National Society for the Prevention of Blindness, 1790 Broadway, New York 19, N. Y.

Reservations should be made in the near future with the Floridian Hotel, 540 West Avenue, Miami Beach, Florida.

Doctor!

Important messages are presented in the advertisements in the MONTHLY regularly, and some ads emphasize services rendered and commodities offered that may be used in your practice—in your office and in the home. Your interest in these encourages advertisers to continue their patronage of your journal. When you send inquiries, wont you say that you saw their advertisement in the VIRGINIA MEDICAL MONTHLY?

Do You Want to Take a Vacation?

Young doctor in resident training has two weeks vacation and will substitute for physician wishing to take a short vacation. Address Dr. James H. Davidson, Watts Hospital, Durham, North Carolina. (*Adv.*)

OBITUARIES

Dr. David C. Morton,

Retired physician of Richmond, died October 13 at the age of 71. He was graduated from the University of Pennsylvania and practiced medicine in Louisville for a number of years before moving to Richmond. Although not engaged in private practice since he moved to Richmond, he was connected with the McGuire Veterans Hospital. His wife survives him.

Dr. Russell B. Bailey,

President-Elect of the West Virginia State Medical Association, died of a coronary occlusion on September 15th. He was fifty-one years of age. Dr. Bailey would have taken office on January 1st as president of the Association.